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MEDICAL JURISPRUDENCE

VOL. II

POISONS

BY

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EDITORS' PREFACE TO FOURTH EDITON.

Since the publication of the third edition of this work in 1873, vast progress has been made in our knowledge of the action of poisons upon the animal economy, and the immense advances which have been made in the domain of organic chemistry have added much to our knowledge of the chemistry of poisons, and have resulted in improved methods of analysis, and, consequently, in the more ready and more certain detection of many of the organic poisons. Therefore it has been found necessary to add so much new material to that portion of the work treating of poisons as to require the assignment of an entire volume to this subject.

It will be seen that much more attention than formerly has been paid to the chemistry of the poisons, the most recent tests and the best methods for their detection in cases of poisoning having been added; the latest advances in our knowledge of the physiological action of the various poisons have also been incorporated, and great care has been taken to collect the more recent cases of poisoning,—especially those illustrating points of medico-legal interest. For assistance in the collection of these cases the editors are much indebted to Charles Harrington, M. D., Assistant in Chemistry in the Harvard Medical School. Thanks are also due to A. Lawrence Mason, M. D., of Boston, for his assistance in the careful revision of the proofs, which was rendered necessary by the unexpected absence in Europe of one of the editors.

An apology may be expected for the long delay in the appearance of this volume on Poisons, but, as will be seen, the vast amount of new material — especially of a technical character — required much patience and a great deal of labor, which could not be deputed to other hands, but must be personally prepared by one or both of the editors.

Robert Amory, M. D.

Edward S. Wood, M. D.

Boston, March, 1884.

EDITORS' PREFACE TO FIFTH EDITION.

In presenting the fifth edition of this work, the knowledge of the action of poisons upon the living organism, as shown by the more recent literature published in Europe and America, has made it necessary for the editors to arrange a complete change from the former classification of the groups of poisons. The older works on the subject of toxicology were inclined to classify poisons in groups indicated by similar symptoms following their administration. Unfortunately, this method of olden time tends to confusion, and, consequently, it has seemed better to classify poisons more in accordance with their chemical and physical relations; an expert, usually familiar with chemistry, more naturally, when investigating the dead body of a person destroyed by poison, pursues a regular order of chemical analysis. His researches, therefore, would be more readily followed under a chemical classification.

Much of the work of the former editors of previous editions has been retained; and some of the text of previous editions has been expunged in compiling the text of the present work. Only the more important judicial cases, some of which were in the text of the previous editions, have been placed in the Appendix to this volume; and those which seemed to have less bearing upon the presentation of the relations of toxicology to judicial inquiries have been omitted. Generally speaking, the cases which have been retained in the Appendix are more for the illustration of the symptoms caused by the poison administered, and of the methods of its detection, or because they are referred to in the text.

The portions of this work which relate especially to chemical research have been thoroughly revised; and obsolete methods for the detection of poisons, which appeared in previous editions, have been replaced by processes which have borne the test of the more recent advances in that science.

In describing the physiological action of the various poisons which appear in this edition, the best of the teachings of the most recent and reliable works on toxicology have been consulted and incorporated; it will be seen from the large number of references in the footnotes

that the compilation of the views of writers on toxicology covers a wide field, explored by experimenters and students in the study of the action of both old and newly discovered articles, not only in their relation to toxicology, but of the results of experiments on the action and effect of poisonous substances during life, and the manner and cause of their producing death. It seems to the editors that this portion of the work is essential for the recognition of the poisons which have brought about the death of victims; this information and knowledge, ranged by the side of the chemical isolation and detection of the poison itself, afford a ready means for the detection of crime, and for the more perfect establishment of the character of the poison used in bringing about the death of a victim.

The editors believe that the book will thus be more serviceable to the professions both of law and medicine, students as well as practitioners; and they sincerely hope that in presenting these various details they will facilitate the course of justice.

Attention is directed to a description of the action of the more recent forms of poison commonly used in the industrial arts, such as carbolic acid, wood alcohol, etc., which, unfortunately, for this reason are accessible to anyone desirous of effecting death by suicide or murder, as also in causing death from accident or negligence. The description of the injury caused to the visual organs by inhalation of the vapor, or from the swallowing of, wood alcohol, has been written by Dr. F. M. Spalding, of Boston, and presents the most modern and important knowledge of these effects of the poisonous action of wood alcohol, the use of which has largely increased both in art and commerce, and as a substitute for ordinary alcohol in the preparation of many substances used in domestic life. The result of this widespread substitution of wood alcohol for ordinary alcohol seems to have caused an increasing number of accidents and suicides.

The attention of the readers of this work is especially called to the new chapters on the so-called ptomain poisoning from foods, and the detection of blood stains, and the differentiation of blood of human from that of the lower animal life.

Former editions have expressed fatal doses in the duodecimal system. The editors of the present edition have translated the doses of poisons mentioned in the text by expressing the corresponding weights and measures in the metrical as well as the duodecimal system. This equivalent is expressed as nearly as possible, although not positively accurate. It should also be observed that degrees of temperature not specifically mentioned as belonging to either Fahrenheit or Centigrade scales refer to the latter.

The use of borax in the preservation of food articles has been so widely known that the bulletin issued by the Agricultural Department of the United States, foreshadowing the results of experiments, to show whether the addition of borax to food articles has an injurious effect upon man, has been added to the Appendix. There has also been introduced in the Appendix the law of the medical examiners of the states of Massachusetts and Connecticut, to which attention is particularly directed.

The editors regret that in order to keep the size of this volume within reasonable limits, and to present the most recent knowledge of toxicology, they have been obliged to cut out much of the previous text which had become classical, in order to cover the most recent and accurate information relating to the action of poisons, and their effects upon organic tissues and organs of the human body; also to provide room for describing the most scientific and accurate methods of isolation and detection by chemical research of the poison used to destroy life.

ROBERT AMORY, M. D.

R. L. EMERSON, M. D.

BOSTON, JANUARY, 1905.

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MEDICAL JURISPRUDENCE.

POISONS.

CHAPTER I.

POISONS IN GENERAL.

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46. Compound poisoning; complication of symptoms due to the combined action of poisons.
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48. Classification of poisons.

1. Definition of poison.— Physicians generally understand by the word "poison" a substance having an inherent deleterious property which renders it, when taken into the system, capable of destroying life. It is difficult, however, to give a definition to the term which will meet the signification attached to it by different classes of persons; for while, in common language, poisons are understood to be those articles only which are deadly in small doses,—as strychnin, prussic acid, arsenic, etc.,—the lawyer and the physician will unite in affixing to it a general meaning similar to that which we have given above. Some substances are habitually classed as poisons, which, according to the popular signification, would have a doubtful claim to be so called, being fatal only in large doses; and every medical practitioner is aware that very many active remedial substances, and some which are not injurious when taken in ordinary amounts, may, in an overdose, produce serious and fatal effects. Moreover, questions may arise as to the applicability of the term to substances which destroy life by mechanical irritation, such as powdered glass, etc., although the administration of such substances to another, with intent to injure, is made a criminal offense in the various penal codes. "Whoever wilfully administers to another, for the purpose of injuring his health, poison or other substance which is capable of injuring the health, shall be punished," etc.[1]

[1] Translated from Das Strafgesetzbuch für das Deutsche Reich, nebst dem Einführungsgesetze vom 31. Mai, 1870.

"Whoever shall administer, or cause to be administered to or taken by any person, any poison, or other destructive thing, with intent to commit murder, shall be guilty of felony."[2] The French law interdicts the wilful administration of any substances "which, without being of a nature to produce death, are injurious to health."[3] In order to avoid the evil of giving too wide or too restricted a meaning to the word "poison," we adopt this definition, which makes no reference to the quantity required to produce a poisonous result, or to the mode in which it was introduced into the system.

Many authors who have written standard treatises on toxicology have attempted to define what constitutes a poison; but definitions as described are often misleading, and have often led to confusion in court trials. One of the recent writers[4] divides poisonous substances into two classes; *viz.*, poisons and corrosives; the former a substance "which, being in solution in or acting upon the blood, may cause death or serious bodily harm; the latter a substance capable of causing death or injury by its chemical action upon a tissue with which it comes in contact."

These authors undertake to illustrate this classification by pointing out that arsenic, which produces insignificant effects by its direct application to the skin, has also a general action upon the whole system, which appears after its passage into the blood; *per contra,* sulphuric acid, applied to the skin, may be absorbed to some extent, but the principal injury is the destruction of the tissues, which "resembles that caused by a burn or a scald." They still further illustrate by suggesting the case of a quarrel between two workmen, during which one is thrown by the other into a vat of sulphuric acid up to his armpits, and so dies from the general destruction of his skin, etc. These authors lay stress upon the fact that the intensity of the action is dependent upon concentration of the corrosive; that of the "true poison" upon its action upon the tissues by absorption into the blood, and that the latter does not cause danger to life in accordance with the degree of its concentration; though exception may be made of those cases where the dilute solution of the poison may be more rapidly absorbed than one which is concentrated, and may therefore act more rapidly. "The intensity of the poisoning depends upon the amount which enters into the blood." This definition of terms lacks positiveness, and is dependent upon certain delimitations.

[2] 24 & 25 Vict. chap. 100, § 11, Aug. 1861, and Taylor's Medical Jurisprudence, 1873, p. 81.

[3] Tardieu, 1875, p. 4.

[4] Witthaus and Becker, Medical Jurisprudence, Am. ed. 1896, p. 43.

We turn now to another high authority, who takes another view of the definitions and distinctions of poisons.[5] In this Manual the statement is made that "the law does not regard the manner in which the substance acts. If it is capable of destroying life or of injuring health, it is of little consequence, so far as the responsibility of a prisoner is concerned, whether its action is of a mechanical or chemical nature, and whether it operates fatally or not." In the Manual from which the above quotation is taken, importance is laid upon the words, "poison or other destructive or noxious things so as to endanger the life of such person, or so as thereby to inflict upon such person any grievous bodily harm." Herin, the editor, calls attention to the ruling of a judge[6] who supported the ingenious contention of the defense "that there must be a distinction between a thing only noxious when given in excess, and a thing which is a recognized poison and is known to be a thing noxious and pernicious in its effects; that unless a thing was noxious in the quantity administered, it cannot be said that there has been a noxious thing administered." Passing over the discussion as to whether cantharides, used in the case from which the above quotation is taken, is primarily a poison because it is placed in the schedule[7] of the English act for regulating the sale of poisons, such as strychnin, prussic acid, etc., we are more concerned with the principle of what constitutes a poison than whether the dose of the substance administered was sufficient to cause death. If the definition of a poison is dependent upon the quantity administered, as an established fact in Medical Jurisprudence, the present list of poisonous substances must be modified by a statement of the dose of a substance, which makes the substance act as a poison. To show how difficult this would be we mention a few well-known articles (not usually considered as poisonous substances) which may become dangerous to life if taken in large amounts: Common table salt, elixir of calisaya bark and iron, and even water when forced into the stomach in large quantities. It would appear to be much better to leave to the competent medical expert, who should be abreast of the times and with the known results of modern technical science, to inform in each case brought for trial or judicial inquiry whether the quantity of the substance supposed to have caused death would ordinarily have been injurious to bodily health, and in what way it may

[5] Manual of Medical Jurisprudence, Alfred S. Taylor, Am. ed. Clark Bell; Lea Brothers, Phila. 1897.

[6] Cockburn, C. J. in *Reg.* v. *Henna*, Cornwall Lent. Ass. 1887.

[7] Criminal law consolidation act, 1861; 24 & 25 Vict. chap. 100, § 11.

have brought about the death. If such a method should be followed, less confusion would result than from a prolonged discussion upon the meaning and play of words and terms. Cases in which a really poisonous substance has been taken with impunity are more rare. In the majority, the immunity is only comparative, the person being affected merely in a less degree than is usual. This important consideration must not be overlooked, for there are many cases recorded in which, for instance, among other deleterious medicines, opium, arsenic, belladonna, digitalis, chloral hydrate, and even strychnin, when the individual has become gradually accustomed to increasing doses, have been taken with impunity in amounts usually considered fatal to mankind. Such cases attest the danger of assigning a deadly action to a poison which has been administered to a person whose peculiar idiosyncrasy (so-called) may resist a dose which to the generality of mankind is considered dangerous or even fatal. Moreover, it has been repeatedly shown that persons who have become habituated to the continuous use of certain drugs, as arsenic eaters, morphin consumers (whether by the mouth or by subcutaneous administration), and the like, require doses far beyond those which would prove fatal to persons in whom the habit does not prevail.

It then follows from what has been above stated that the use of definite words or phrases to restrict or comprise a schedule of poisons as a medico-legal requirement is in general unadvisable. The medical expert should be well qualified to explain clearly to the court in what manner the substance in a given case may, or usually does, destroy life, and the amount of it required to be in contact with the tissues the life and functions of which when alive are shown by him to have been destroyed. A scheme classifying substances of similar nature, in producing similar effects upon the animal tissues, may help him to expound the morbid action more clearly to the satisfaction of a court of inquiry.

2. Conversion of harmless substances.—The idiosyncrasy which converts a harmless substance into a poisonous agent is very frequently observed in the case of articles of food. Thus mussels, fish, pork, and mutton have frequently given rise to all the symptoms of irritant poisoning. It should be remembered, however, that the symptoms in these cases may result as well from the mechanical irritation of the food,—too large a meal having been taken,—or from the fact of its being in a condition unfit for use; while it may be seen, farther on in this treatise, that some meats become poisonous because they are infested with animal parasites, or from commencing putrefaction.

3. **Food poisoning.**— In late years the matter of food poisoning has been brought very prominently into view. Irritant foods and food poisoning, on account of organic animal and vegetable fermentation, may cause serious gastric, as well as intestinal, irritation, even causing an acute inflammation of the whole alimentary canal; and there may ensue a still further conversion of these altered food products into poisonous substances, by a well-known physico-chemical process, called ptomains, leukomains, etc., and these may be absorbed into the circulation of blood, and cause peculiar symptoms of intoxication by the secondary poisonous products. The production of these ptomains is generally attributed to bacterial growths within the tissues of the living or dead body. Many of the ptomains give similar reactions, with difficulty distinguishable from the reactions to chemical tests given in the presence of the alkaloids. The medical expert should, however, be able to distinguish by the aid of the microscope and culture fluids, and to study the development and propagation of the bacteria which accompany the production of these ferments. The older name of "ptomain" has more recently been superseded by the use of the word "toxin." The discussion of this agent, which is a veritable poison, will be found in the sixth chapter, which will be limited to Food Poisons.

Formerly the origin of ptomains was supposed to be dead matter,— matter of animal material only; but more recently this theory and the restriction of the origin of alkaloids to vegetable matter has been abandoned by the physiological chemists of advanced science. We take the liberty of referring to a recent toxicologist, who has presented an epitome of the status of this discovery (Dr. Luff, Vol. I., pp. 397 *et seq.*): "The word 'ptomain' is derived from πτῶμα, a dead body. Ptomains, also known as cadaveric alkaloids, are alkaloids produced by the decomposition of animal substances. The term 'ptomain' was at first restricted to alkaloids produced by cadaveric decomposition, but now it is also employed to designate alkaloids of animal origin formed during life as a result of chemical changes induced by some agency acting within the organism.

"The term 'leukomain' has recently been introduced to particularize the animal alkaloids formed during life from those produced by decomposition of dead animal matter; but it would be preferable for the terms 'ptomains' and 'leukomains' to be abandoned, and to class these bases of animal origin in one category as animal alkaloids.

"It is now well known that the power of manufacturing alkaloids

is not restricted to plants, but is shared by animal organisms." After enumerating a number of these alkaloids which have been discovered by recent physiological chemists, Dr. Luff continues by stating as follows: "Since then, Gautier and Brieger have made extensive researches, resulting in the discovery of several animal alkaloids. . . . In 1882 Bouchard demonstrated that, not only were alkaloids present in appreciable quantities in normal urines, but that they augmented notably in the course of certain maladies,—typhoid fever for instance. These results have been confirmed by Lepine and Aubert, and by the author, who has extracted from the urines of typhoid and scarlet fever patients ptomains which are present in greatest amount during the height of the fever, but which diminish as the fever subsides."

"Animal alkaloids are also a necessary product of vital physiological processes, and have been extracted from the secretions of living beings and from animal tissues." Dr. Luff then presents a list of the principal ptomains extracted from putrefying animal matter.

It is necessary in this opening chapter to call attention to the train of serious symptoms which are often observed to follow the ingestion of ergot (the growth of fungi on rye and other cereals). Mention should also be made of the serious symptoms which are sometimes observed to follow the eating of mussels which have been taken out of stagnant water, or the food taken from heaps of unwholesome surroundings; these may cause gastro intestinal disturbance, even when not taking into account the more serious spread of a disease germ which disseminates typhoid fever and other filth diseases. The eating of unwholsome food may often give rise to suspicion of an active poison, because the symptoms which follow appear so serious.

Dr. Vivian Poore writes: "In regard to this question of ptomains, there is an interesting thing recorded by Professor Victor Vaughan, to which I have previously alluded, and to which I will allude again. It was found by him that tissues impregnated with arsenic, if allowed to decompose, gave off a garlicky odor; and the arsenic disappeared. . . . In the year 1888 there was at Middleborough an epidemic of pneumonia, and this epidemic was investigated by Dr. Ballard. He found that the idea was prevalent throughout Middleborough that the pneumonia was largely among those who had eaten a particular form of bacon. That was quite a new departure, and Ballard, with his open mind, investigated the matter seriously. He found that there was a great deal of truth in this, and, when he had collected all his cases of pneumonia, he found a large proportion of them had partaken

of a particular form of bacon. He found that the workingmen of Middleborough had the idea, which is probably common, and I should not be surprised if it is tolerably true, that if you want to get the most good out of meat it must not be overcooked, it must be underdone. He found that Middleborough artisans had been in the habit of eating this bacon very underdone indeed, almost raw, just warmed before the fire. He found that this bacon was made from American pork rapidly cured by a process of injection, or something of that sort. . . . Ballard found that this pork was being prepared in an unwholesome place in a factory in which there was a sewer grating. He scraped the fat off the grating, and sent some of the fat and some of the pork up to London to Dr. Klein, who found not the ordinary diplococcus of pneumonia, but another one which was capable of giving pneumonia, and which produced an epidemic among rabbits and guinea pigs and killed a great number of them. . . . There is some doubt (pneumonia caused by eating pork), and I notice that in recent writings this particular instance has been dropped. . . . Nevertheless, I think Ballard's figures are incontrovertible, that pneumonia did occur in excessive proportions among the pork eaters, and a dangerous bacillus was found on this pork,—especially in the fat which was scraped off the sewer grating."[8] The above case is instructive, even if we do not go to the lengths to which our author follows it; we should, however, suggest that in our opinion it is rather a matter of regret that rabbits and guinea pigs were used to control the results of the experiment, because these animals—especially when confined in close cover—are so liable to pulmonary diseases. The case quoted is interesting on account of the suspicion involved, and as a group of cases affected from a common cause,—all partaking of the injurious food.

There is another source of food poisoning which should not be overlooked, and which has become historical as an incident in our late Spanish-American war; we refer to meats sold in tin cans. If in preparing these canned meats care is not taken to drive out the atmospheric air by thoroughly boiling the liquid contained in them, there may occur a decomposition which will produce an injurious substance; this may not necessarily cause death, though it does commonly produce serious gastro intestinal disturbances, such as diarrhea and colicky pains, which may simulate a diseased condition. It was formerly supposed that this train of symptoms was due to the action

[8] A treatise on Medical Jurisprudence; Longmans, Green, & Co. N. Y. 1901.

of the zinc and lead compounds used in soldering the cans, but it is more likely caused by products of fermentative processes, which are favored by the contained air not having been driven out by long-continued boiling. It should be remembered that these deleterious substances cannot always readily be recognized by taste and smell. The medical expert should be qualified to recognize the micro-organisms by the aid of the microscope and their propagation in specially prepared cultures (such as gelatine, soup, bouillon, etc.).

It is well to note here that in the present knowledge of physiology the small intestine is recognized as the great absorbent organ of the digestive tract, and the large intestine as an eliminating organ; hence when diarrhea exists as a symptom after the ingestion of a poisonous substance, from its irritating action on the mucous coating of the alimentary canal, the diarrheal discharge favors the elimination of the poison; yet this very process of irritation may leave, as a result, points of inflammation.

It must be recognized that the quantity of any poison which may cause injury to the living tissues in the human body and destroy their functions of life is dependent, not so much upon the amount of the substance which is administered by the mouth, as by the amount which is retained in the system and in contact with these living tissues. This is also dependent on the rapidity of the elimination after the ingestion. If the substance is removed very rapidly by the process of diarrhea, and without causing serious injury to the tissue of the alimentary canal, an insufficient amount may remain to be absorbed by the blood to produce a poisonous action upon the general system. The medical expert should ascertain how much was retained in the system, and whether this was an amount sufficient to cause serious injury. He should remember that the amount found in the large intestine may not do any more harm than the local action due to its direct contact with that organ of elimination.

4. **Disease preventing effect of poison.**—Disease also has sometimes the effect of rendering the system tolerant of substances which would be poisonous in the same doses in a healthy state of the system. In acute alcoholism, or alcoholic intoxication, the blood and tissues may be so loaded with alcohol as to interfere wholly or partially with the absorption of a poisonous agent. This fact is so well known to the medical profession, that the tolerance of chloral hydrate, belladonna, digitalis,[9] and opium leads physicians to give larger and more frequent

[9] Medical Times and Gazette, Aug. 23, 1873, p. 205.

doses in delirium tremens than would seem to the inexperienced to be safe. In consequence of this knowledge, fatal accidents have occasionally occurred[10] from the inconsiderate repetition of large doses to inebriate patients, who, later, might have eliminated the alcohol and recovered from its intoxicating effects. This same observation and explanation may be true in certain diseases, where, apparently, the blood contains an organic poison due to functional or organic disease of the kidneys, and thus seems to offer a limited opposition to the absorption of a toxic agent.[11] During the active stage of severe inflammatory and febrile diseases, mercury may be given in large and repeated doses without producing salivation. On the other hand, certain diseases render the body more susceptible to the actions of poisons. Illustrations of this are presented by the aggravation, or the ready production, of cerebral symptoms after the use of small doses of narcotics, in those who have a predisposition to cerebral congestion or apoplexy, and by the extreme facility with which salivation follows the administration of mercury in persons affected with certain forms of disorganization of the kidneys. The use of iodid of potassium after mercurial preparations is known to favor the development of the mercurial cachexia.

These facts tend to show that the activity of a poison depends largely upon the readiness with which the poison is absorbed in any individual case. It is unnecessary to state here all of the circumstances which govern the rapidity of absorption. This discussion should be left for each case as it arises, and be elucidated from the testimony of a competent medical expert, it being remembered that no general rule can be precisely laid down (see p. 40), and that certain diseases or abnormal conditions of the system may facilitate, while others may retard, the action of a poison.

5. **Evidence of poisoning.**—Medical evidence in cases of suspected poisoning is derived from several sources; to wit, the symptoms, the post-mortem appearances, chemical analysis, and experiments on animals; these are also called the clinical, anatomical, chemical, and physiological signs of poisoning. In addition we have the moral evidence derived from nonmedical facts, concerning which the physician, as well as other persons immediately connected with the case, must

[10] Irish Hospital Gazette, 1873; Year Book of Therapeutics and Pharmacy, 1872, p. 254; Deutches Archiv. Bd. VIII. Jan. 1871, quoted in The Practitioner for April, 1871, p. 243; Lancet. Feb. 1, 1879, p. 147; Br. Med. Jour. Aug. 29, 1868; Lancet, Aug. 27, 1870; De l'Influence des Liquides Alcooliques, Dubois (Thèse de Paris, 1870).

[11] Lo Sperimentale, Fasc. 9, 1873, quoted in The Practitioner, Feb. 1873, p. 132.

testify. In order to establish the fact of poisoning with certainty, everything, of however little importance it may seem to be at the time, which occurs during the life of the patient, must be observed and accurately noted. Especially should the physician in attendance upon any case, where there is the slightest suspicion of poisoning, take charge of everything which may by any possibility throw light upon the cause of the illness, such as all vomitus and excreta (urine and feces), bottles of medicine, powders, and any article of food or drink taken previous to the illness, if such can be obtained; all of these substances should be carefully preserved by being placed in clean glass vessels (ordinary glass preserve jars will answer the purpose) by the physician himself, sealed, and kept under lock and key until wanted. Of the greatest importance is the collection of the vomitus and urine during the life of the patient, if possible, for the detection of the nature of the poison by chemical analysis of these fluids.

6. Statistics of poisoning.— The statistics of poisoning show that the kind of poison most frequently used varies very greatly according to circumstances, such as the ease with which poisons can be obtained by the laity, the advancement of popular knowledge concerning the poisonous action of substances used in the arts and manufactures, etc. Hence the variation in frequency with which the different poisons are used in different countries and in different periods. Thus cases of criminal poisoning by phosphorus were unknown previous to the year 1850 on account of the ignorance of people generally as to its poisonous effects.

In Prussia during the years 1869 to 1873, of 32,613 deaths, 1,454 were due to poisoning, as follows:[12]

Poisonous gases	1007
Poisonous berries and fungi	62
Poison acids	40
Arsenic, phosphorus, and other poisons	57
Alcohol	288

In England and Wales, during the five years 1863–1867, the cases of poisoning were as follows:[13]

Opium—laudanum, syrup of poppies, and Godfrey's cordial	482
Opium and its compounds	114

[12] A. V. Fürcks, Rückblick auf die Bewegung der Bevölkerung im Preussischen Staate während des Zeitraumes vom Jahre 1816 zum Jahre 1874. From Maschka's Handbuch der gerichtliche Medecin, II. p. 21.

[13] Taylor on Poisons, 1875, p. 179.

Morphin	32
Prussic acid and cyanid of potassium	151
Essential oil of almonds	31
Arsenic	83
Oxalic acid	66
Strychnin and vermin killer	61
Mercury (compounds of)	58
Sulphuric acid	53
Nitric acid	16
Hydrochloric acid	8
Carbolic acid	5
Alcohol	35
Phosphorus	15
Ammonia	11
Chlorid of zinc	8
Mussels	8
Fungi	6
Aconite	6
Belladonna	6
Chlorodyne	4
Turpentine	3
Colchicum	3
Niter	3
Sulphate of copper	3
Cantharides	2
	1273

Mr. Alexander W. Blyth, in a recent publication, gives a list of cases of individuals who have died from poisons in England and Wales,—6,616 persons, as shown in the following table:

TABLE A.

Deaths from Poison in England and Wales During the Ten Years 1883–1892.

	Accident or negligence.		Suicide.		Murder.		Total.	
Metals:	M.	F.	M.	F.	M.	F.	M.	F.
Arsenic	37	14	37	20	1	1	75	35
Antimony	3	....	1	2	....	...	4	2
Copper	4	1	2	1	....	...	6	2
Lead	831	209	1	2	....	...	832	211
Silver nitrate	1	....	...	...	....	...	1	...
Zinc chlorid (or sulphate)	7	....	4	...	....	..	11	...
Mercury	22	11	16	8	2	1	40	20
Chromic acid	1	....	....	...	....	...	1	...
Iron perchlorid	...	....	....	1	....	...	....	1

TABLE A.—*Continued.*

Deaths from Poison in England and Wales During the Ten Years 1883–1892.

	Accident or negligence.		Suicide.		Murder.		Total.	
Alkaline earths:	M.	F.	M.	F.	M.	F.	M.	F.
Lime	2	....	....	1	....	....	2	1
Barium chlorid	1	....	....	...	....	....	1	....
The alkalies and their salts:								
Ammonia	39	25	18	16	....	....	57	41
Caustic soda	3	4	....	1	....	....	3	5
Caustic potash	8	10	1	....	....	....	9	10
Potassic chlorate	1	....	....	....	....	....	1	....
Potassic bichromate	2	2	7	3	....	....	9	5
Potassic bromid	1	....	....	....	....	....	1	....
Potassic binoxalate (sorrel)	1	3	1	4	....	....	2	7
Acids:								
Sulphuric acid	30	9	29	24	1	....	60	33
Nitric acid	18	7	18	9	....	....	36	16
Hydrochloric acid	48	18	83	55	....	....	131	73
Oxalic acid	17	6	114	86	....	....	131	92
Tartaric acid	...	1	....	....	....	....	....	1
Acetic acid	4	3	....	2	....	....	4	5
Carbolic acid	169	101	219	271	....	1	388	373
Hydrofluoric acid	...	....	....	1	....	....	....	1
Phosphorus (including lucifer matches)	24	47	28	56	....	....	52	103
Iodin	6	7	1	1	....	....	7	8
Volatile liquids:								
Paraffin (petroleum)	9	2	1	....	....	....	10	2
Benzolin	3	2	....	1	....	....	3	3
Naphtha	1	....	....	....	....	....	1	....
Carbon bisulphid	...	....	1	....	....	....	1	....
Turpentine	5	1	....	3	....	....	5	4
Methylated spirits	...	2	1	2	....	....	1	4
Alcohol	81	24	1	2	....	....	82	26
Chloroform	57	41	9	5	1	....	67	46
Ether	5	2	....	....	....	....	5	2
Spiritus ætheris nitrosi	1	....	....	....	....	....	1	....
Anesthetic (kind not stated)	4	3	....	....	....	....	4	3
Oil of juniper	1	....	....	....	....	....	1	....
Opiates and narcotics:								
Opium, laudanum, morphia	503	373	330	167	4	2	837	542
Soothing syrup, paregoric, etc.	18	22	2	3	....	....	20	25

TABLE A.—*Continued.*

Deaths from Poison in England and Wales During the Ten Years 1883–1892.

	Accident or negligence.		Suicide.		Murder.		Total.	
Opiates and narcotics:	M.	F.	M.	F.	M.	F.	M.	F.
Chlorodyne	56	30	8	8	....	....	64	38
Chloral	89	22	14	1	1	....	104	23
Cyanids:								
Prussic acid and oil of almonds	17	11	203	19	2	8	222	38
Potassium cyanid	19	21	100	22	3	1	122	44
Alkaloids:								
Strychnin and nux vomica	22	21	65	85	4	4	91	110
Vermin killer	2	6	49	69	1	....	52	75
Atropin	2	....	1	....	....	....	3	....
Belladonna	36	20	11	9	....	....	47	29
Aconite	19	21	9	10	....	....	28	31
Ipecacuanha	1	1	....	....	....	....	1	1
Cocain	3	....	....	....	....	....	3	....
Miscellaneous:								
Antipyrin	1	....	....	....	....	....	1	....
Cantharides	1	....	....	1	....	....	1	1
Camphorated oil	1	....	....	....	....	....	1	....
Croton oil	1	....	....	....	....	....	1	....
Cayenne pepper	1	....	....	....	....	....	1	....
Syrup of rhubarb	1	....	....	....	....	....	1	....
Colchicum	2	....	....	....	....	....	2	....
Hemlock	3	1	....	....	....	....	3	1
Water hemlock	5	6	....	....	....	....	5	6
Colocynth	...	2	....	....	....	....	....	2
Castor oil seeds	1	1	....	....	....	....	1	1
Laburnum seeds	2	1	....	....	....	....	2	1
Thorn apple	1	....	....	....	....	....	1	....
Yew leaves or berries	3	2	....	....	....	....	3	2
Crowfoot	...	1	....	....	....	....	....	1
Whin flower	1	....	....	....	....	....	1	....
Pennyroyal	...	1	....	....	....	....	....	1
Meadow crowfoot	...	1	....	....	....	....	....	1
Arum seeds	...	1	....	....	....	....	....	1
Bitter aloes	...	1	....	1	....	....	....	2
Cocculus indicus	...	....	1	....	....	....	1	....
Horsechestnut	...	1	....	....	....	....	....	1
Creasote	1	....	....	....	....	....	1	....
Spirits of tar (oil of tar)	2	1	....	....	....	....	2	1

TABLE A.—*Continued.*

Deaths from Poison in England and Wales During the Ten Years 1883–1892.

	Accident or negligence.		Suicide.		Murder.		Total.	
Miscellaneous:	M.	F.	M.	F.	M.	F.	M.	F.
Nitroglycerin	1	....	....	....	....	....	1	....
Camphor	...	1	....	....	....	....	....	1
Tobacco	4	....	1	....	....	....	5	....
Lobelia	1	....	....	....	....	....	1	....
Fungi	13	10	....	....	....	....	13	10
Poisonous weeds	2	....	....	....	....	....	2	....
Hellebores	...	....	1	1	....	....	1	1
Kind not stated	216	158	256	167	3	1	475	326
Total	2,498	1,292	1,644	1,140	23	19	4,165	2,551

Under the medical examiner statute in Massachusetts the official medical examiner is required to report to the secretary of state each case of suspected death by violence which he is called to "view." We have made the following tables of every case of death after the taking of a poison, which have been collated from the original reports of theses medical examiners:—

Table Exhibiting Cases of Death from Poisons, Compiled from Original Returns from Massachusetts Medical Examiners to the Secretary of State, 1877–1902, inclusive.

NONCRIMINAL (ACCIDENT OR NEGLIGENCE).

	'77	'78	'79	'80	'81	'82	'83	'84	'85	'86	'87	'88	'89	'90	'91	'92	'93	'94	'95	'96	'97	'98	'99	'00	'01	'02	Total
Arsenic, compounds	...	1	...	2	...	...	1	...	2	2	...	...	2	...	...	4	...	...	2	4	2	1	...	4	...	2	29
Opium preparations	...	...	3	1	4	...	5	1	3	...	...	4	2	1	1	3	2	7	6	6	2	3	7	...	5	8	74
Strychnin	...	...	...	...	...	1	...	...	...	...	...	1	1	1	...	...	...	...	2	2	1	2	1	2	3	2	19
Chloral hydrate	...	...	1	...	3	3	1	1	...	...	...	...	...	...	...	...	...	...	1	2	1	1	...	...	...	...	14
Mercury bichlorid	...	...	...	...	...	...	...	...	1	1	...	1	...	...	...	...	...	...	2	...	...	1	1	...	3	...	10
Phosphorus	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	2	2	...	1	...	...	1	...	6
Carbolic acid	...	...	...	...	1	...	1	2	1	...	...	...	1	1	...	...	1	3	2	...	1	2	3	...	...	2	21
Prussic acid, etc.	...	...	...	...	...	...	...	...	1	...	...	1	...	...	...	...	3	...	...	...	...	...	...	...	...	...	5
Muriatic acid	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	1
Methyl alcohol	...	...	...	...	1	1	...	...	...	...	...	...	...	1	1	...	...	...	...	...	...	...	...	...	6	...	10
Chloroform	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	1	3	1	...	...	...	...	...	...	...	6
Aconite	...	...	...	...	...	1	...	1	...	1	1	1	...	...	...	...	...	...	...	...	...	1	...	...	...	...	6
Oxalic acid	...	...	...	...	...	...	...	...	...	2	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	3
Ammonia, including washing fluid	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	1	...	2	...	1	...	...	5
Digitalis	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	1	3
Cedar, oil of	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	1
Wintergreen, oil of	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	6	...	...	1	...	...	...	...	...	8
Tansy, oil of	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	2
Lobelia	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
Canned corn	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
Podophyllum	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
Castanea nuts	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	1
Wild parsnip root	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	2	...	...	1	...	...	...	...	...	3
Soapine (potash)	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	1
Embalming fluid	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
Nitric acid	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	1	...	...	...	2
Stramonium seeds	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	1
Lead	...	...	...	...	...	...	1	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	2

Coal dust	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	1
"Acetanilid"	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	1	...	...	...	2
Caustic potash	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	1
Narcotic comp'd	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
"Fly paper"	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	2	...	2
White hellebore	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	1	2
Water hemlock (Cicuta mac.)	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	1
Rattlesnake bite	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	4	...	4
(Not stated)	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
Total	...	1	5	3	9	6	11	8	8	8	1	8	6	5	4	7	7	23	19	18	11	16	15	9	26	17	251

CRIMINAL (HOMICIDE).

	'77	'78	'79	'80	'81	'82	'83	'84	'85	'86	'87	'88	'89	'90	'91	'82	'93	'94	'95	'96	'97	'98	'99	'00	'01	'02	Total
Alcohol (death twelve hours)	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
Arsenic compounds of	...	1	...	2	...	...	...	...	...	5	...	...	1	...	1	...	...	...	...	...	...	...	...	...	...	...	10
Opium preparations	...	...	...	...	...	...	...	...	...	1	2	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	4
Strychnin	...	...	...	...	...	...	...	1	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	2
Muriatic acid and soldering salts	...	...	...	...	...	...	...	...	...	...	1	...	1	...	1	...	...	...	...	...	...	...	...	...	...	...	3
Chloral hydrate	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
Turpentine	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
Tansy, oil of	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
Cedar, oil of	...	1	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	2
Pennyroyal, oil of	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
Kerosene oil (external)	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
Prussic acid and cyanids	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	2	...	...	...	...	...	...	...	...	...	2
Methyl alcohol (wood)	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
(Not stated)	...	...	...	...	...	...	...	2	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	2
Total	...	4	...	2	...	...	2	3	1	7	4	2	2	...	3	...	2	...	...	...	...	...	...	...	...	...	32

NONCRIMINAL (SUICIDE).

	'77	'78	'79	'80	'81	'82	'83	'84	'85	'86	'87	'88	'89	'90	'91	'92	'93	'94	'95	'96	'97	'98	'99	'00	'01	'02	Total
Arsenic, compounds of	2	2	6	3	2	2	3	16	13	13	33	22	26	15	17	20	20	18	16	29	22	23	16	14	13	14	380
Opium preparations	4	4	5	5	1	2	8	10	14	6	10	16	17	23	21	27	21	21	20	27	20	16	19	13	14	13	357
Strychnin	...	...	...	...	1	...	...	...	2	2	2	1	2	4	...	1	1	1	1	1	...	1	...	3	3	1	27
Zinc, salts of (soldering)	...	...	...	...	...	...	...	...	...	...	1	...	1	...	...	1	...	...	...	...	1	2	...	2	1	1	10
Chloral hydrate .	...	...	1	...	...	1	1	...	1	...	...	3	3	2	2	5	4	...	1	2	3	1	1	3	...	...	34
Chloral with hyoscyamus	...	...	...	...	...	1	1	...	...	...	...	...	...	...	2	...	...	...	...	...	...	...	...	...	...	...	4
Chloral with morphin, etc.	...	...	...	...	...	...	1	1	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	3
Creasote	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	1	2
Hemlock, oil of ..	...	...	...	...	...	...	...	...	...	...	...	1	2	...	...	...	...	...	...	...	...	...	...	...	...	...	3
Mustard, oil of ..	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	1
Mercury bichlorid (cor. sub'ate) .	...	...	1	...	1	...	1	2	...	...	1	1	1	...	1	...	2	...	1	4	1	2	2	2	...	3	26
Pennyroyal, oil of	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	1	...	...	1	1	...	...	4
Phosphorus (and matches)	...	...	...	...	1	...	2	...	1	1	2	...	2	...	...	...	...	...	...	1	...	1	...	...	...	...	11
Carbolic acid ...	...	...	...	...	...	...	...	3	2	...	...	...	4	3	1	4	3	3	2	6	15	25	20	20	20	24	155
Cresoline	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	1	...	...	2
Prussic acid, cyanids	...	...	1	2	...	...	1	...	4	2	1	2	2	...	1	1	4	4	4	4	6	7	7	4	4	7	68
Muriatic acid ...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	1	...	...	...	...	1	...	3
Methyl alcohol (wood)	...	...	...	...	...	...	...	...	...	...	...	...	...	1	2	...	1	1	1	...	1	2	6	8	...	4	27
Chloroform	...	...	...	...	...	...	...	...	3	...	2	...	...	2	4	1	1	...	4	2	3	1	3	5	1	1	33
Turpentine, shellac, etc.	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	1	...	...	...	...	...	2
Aconite	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
Oxalic acid	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
Ammonia and washing fluid .	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
Rue	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
(Not stated)	...	...	...	...	...	...	2	...	...	...	1	3	...	...	...	...	...	...	...	...	...	...	...	...	...	...	6
Total	6	7	14	11	6	6	20	32	41	26	54	49	60	50	51	62	58	48	56	78	73	81	76	77	57	69	1162

The total number of deaths from poisoning, as shown in the foregoing three classes, during twenty-five years ending with the year 1902, amounts to 1,439, or about 57 each year of the population of Massachusetts (2,522,145, census of 1900). This would indicate, for each year, one death by poison to 450,000 inhabitants. If we compare this ratio with that presented by Mr. Blyth, and figure the total deaths shown in his table for a period of ten years with the census for England and Wales (1890; 29,000,525), we find there was one death in each year to 440,000 inhabitants, it being noted that the population is nearly eleven and a half times greater. It would hardly be fair to leave this comparison without the remark that the strict requirements of the Massachusetts statutes compel very full and complete returns of all deaths by violence, by means of a large and able body of carefully selected physicians as medical examiners, while in England the older form of coroner law prevails.

In Massachusetts the state medical examiners reported, out of each 2,976 supposed deaths by violence viewed by them, that 45 were deaths from poisoning.[14]

Of late years there have been reported quite a number of cases in which death, by suicide and by accident or negligence, has occurred by the swallowing of carbolic acid; in the first instance, because it can be so readily obtained; and, in the second instance, because of its peculiar resemblance, both in odor and taste, to whisky.

Tardieu[15] gives the statistics in France for twenty-one years, 1851–1871, as follows:—

[14] Transactions of Massachusetts Medico-Legal Society for 1878–81, Cambridge Riverside Press.

[15] Etude sur l'Empoisonnement, 1875, p. 164.

	'51	'52	'53	'54	'55	'56	'57	'58	'59	'60	'61	'62	63	'64	'65	'66	'67	'68	'69	'70	'71	Total
Arsenic	35	25	33	25	42	14	18	9	9	3	14	5	8	7	6	4	4	8	14	2	2	287
Phosphorus	13	3	4	12	21	14	23	20	16	15	13	16	5	25	12	6	18	11	11	4	3	267
Sulphate of copper	3	5	10	8	4	2	8	5	15	5	4	8	2	9	4	7	7	2	2	5	5	120
Verdigris	2	3	10	4	6	3	1	...	...	2	2	...	...	...	1	1	3	1	...	...	...	39
Sulphuric acid	1	1	2	1	2	7	1	4	4	4	1	2	...	...	...	...	2	...	3	1	...	36
Cantharides	4	1	3	3	...	6	1	2	2	...	1	...	...	3	...	1	2	...	1	...	5	35
Opium	...	...	...	1	...	...	...	...	1	1	1	2	...	...	...	...	...	...	...	...	...	6
Hellebore	...	...	...	...	...	...	...	...	...	...	2	...	...	...	...	1	...	...	...	...	...	3
Tartar emetic	1	...	...	...	...	...	1	1	...	...	...	1	...	...	...	...	...	...	...	...	...	4
Sulphate of iron	1	...	...	...	...	...	2	...	...	...	...	1	...	...	...	...	...	...	2	...	...	6
Nitric acid	1	...	1	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	3
Ammonia	...	...	1	...	...	...	...	...	1	...	1	...	1	...	...	...	...	...	...	...	...	4
Mercury	...	...	1	...	1	...	...	...	...	1	...	...	1	...	...	3	...	1	...	...	...	8
Datura	...	...	3	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	3
Nux vomica	...	...	...	...	2	...	...	...	...	1	...	...	...	...	...	...	1	...	...	...	1	5
Hydrochloric acid	...	...	...	1	...	...	...	...	1	...	...	...	1	2	...	3	...	...	...	...	...	8
Potash	1	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	2
Acetate of lead	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	0
Carbonic acid gas	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	1
Broom seeds	...	...	1	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	2
Colchicum	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
Mushrooms	...	...	1	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	2
Euphorbia	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
Baume Floraventi	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
Aqua sedativa / Eau sedative	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	1
Belladonna	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
Powdered glass	...	1	...	...	...	1	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	3
Strychnin	...	...	...	...	...	...	...	...	...	...	...	...	...	3	1	1	1	...	1	...	...	7
Digitalin	...	...	...	...	...	...	...	...	...	...	...	...	...	2	...	...	...	...	...	...	...	2
Nicotin	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	1	...	...	2
Prussic acid	...	...	...	...	...	...	...	...	...	...	...	...	...	1	1	...	...	...	...	...	...	2
Laudanum	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	1	2
Antimony	...	...	...	...	...	...	...	...	...	...	...	...	...	1	1	...	...	...	...	...	...	2
Croton oil	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	1	2
Sulphate of zinc	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	1
Tincture of iodin	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	1
Eau de Javelle	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	1
Sulphuric ether	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	0
Cyanid of potassium	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	2	...	...	...	2
Total	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	873

Professor Poore has presented, in his recently published lectures on toxicology, from which we have previously quoted, a list of the more common poisons used in England:—

Acids.
- Sulphuric (oil of vitriol).
- Nitric (aqua fortis).
- Hydrochloric (spirits of salt).
- Oxalic acid (salts of sorrel).
- Carbolic acid (phenol).
- Hydrocyanic acid, and cyanids (prussic acid).

Alkalies.
- Ammonia.
- Potash (caustic).
- Soda (caustic).

Aconite (aconitin).
Alcohol.
Anilin (antifebrin, exalgin, and the like proprietary drugs).
Antimony (tartar emetic).
- (wine of).

Arsenic (in acute poisoning).
- arsenious acid.
- white arsenic.
- cacodylic compounds (organic arsenical compounds).

Belladonna (atropin).
- (deadly nightshade).

Chloral hydrate.
Chlorin gas.
Chlorodyne.
Chloroform.
Cocain.
Colchicum.
Conium (hemlock).
- (spotted hemlock).

Copper.
Corrosive sublimate (bichlorid of mercury).
Digitalis (foxglove).
Henbane.
Laurel water (a solution of prussic acid).
Lead (acute poisoning).
Lunar caustic (silver nitrate).
Morphia, acute (by mouth or hypodermically).
Nightshade (solanum dulcamara).
- (solanum nigrum).

Nitrobenzene (nitrobenzol).
- (artificial oil of bitter almonds).

Nux vomica.
Opium. (See morphia.)
Phosphorus.
Physostigma (calabar bean).

Pilocarpin.
Savin.
Strychnin. (See nux vomica.)
Tobacco.
Turpentine.

7. Symptoms, in general.—It is but rarely that some knowledge of the symptoms preceding death is not obtained, even where the mode of their invasion has been unobserved. Occasionally, indeed, persons are found dead from the effects of poisoning, of the precise manner of whose death nothing can be learned, the suspicion of poisoning arising only in consequence of the finding of the vial from which the poison had been taken, or other circumstantial evidence of a similar character. Such are, in general, cases of self-destruction; the unfortunate victim of misfortune or excess having designedly withdrawn himself from observation, with a view of being undisturbed in his purpose.

A notable instance of such a case of deliberate suicidal poisoning, in which the circumstances were so carefully planned as to avert suspicion of poisoning, is personally known to the writer. The details of the tragedy may be briefly summarized. A man in perfect health, on a very hot day in summer, took a private room in a hotel, and, complaining of a violent headache, sent the messenger who answered his call to an apothecary shop for some camphor spirits, which he poured upon a handkerchief and bound on his forehead, at the same time dismissing the messenger. He was found dead several hours afterwards, and his death was attributed to a congestion of the brain brought on by the heat of the weather. Suspicions of suicide being aroused later, it was found that he had poisoned himself with prussic acid, and disguised its smell by the external application of camphor. No bottle or paper was found in the room or about the person to point in the slightest degree to poisoning as the cause of death, yet chemical examination of the stomach showed a large amount of prussic acid. The post-mortem appearances were, of course, those of general venous congestion, found in cases of prussic acid as well as some other forms of poisoning, especially cerebral congestion.

In most cases of accidental and homicidal poisoning, some knowledge is acquired, either directly or indirectly, of the nature and progress of the symptoms. With few exceptions, however, medical aid is sought before death has occurred, and the direct testimony of the physician can thus be obtained. The history of the symptoms

should in all cases be made in great detail, as these form a very important aid in the detection of criminal poisoning.

8. Symptoms as dependent upon avenue through which poison is administered.—The first effect of a drug would, of course, be its local action, and this should be looked for upon any of the avenues through which poisons may be administered. A corrosive poison, if taken by the mouth, would show its local effects upon the face, lips, etc.; but if the suspected poison be not a corrosive or a local irritant, the first condition of the intoxication (poisoning) is its absorption, and if this absorption does not occur, fortunately there are no poisonous effects to be noted; for example, the absorption of arsenious acid may be delayed or prevented by the ingestion of some albuminous substances, such as milk, thus locking up the poison in a mass, which cannot, without subsequent digestion, be received into the circulation.

There are several ways by which poisonous drugs may be introduced into the system. The principal of these are the mucous membranes, the lungs, the cellular tissue, and inflamed serous membranes. Whatever may be the way by which the agent is introduced, science possesses the means of recognizing deleterious substances, whether in the blood, the secreted fluids, or in the tissues themselves.

The careful researches of physiologists (Prof. Cl. Bernard and Prof. Gubler among others) of modern times show clearly that any mucous surface can absorb medicaments; briefly stated, these avenues for the administration may be enumerated in the order of rapidity as follows: The surface of the lungs, stomach, intestines, mouth, nose, eyes, tear passages, rectum, vagina, uterus, bladder (as in case of atropin), and prepuce. The presence of food in the stomach and bowels interferes also with the rapid absorption of a drug or poison, because the presence of alimentary substances separates the drug from contact with the absorbing surface. The lungs will absorb not only gases and volatile poisons, such as sulphureted hydrogen, coal gas, ether, chloroform, prussic acid, etc., but also the watery vapors holding in suspension particles of drugs or poisons, and even dust which is suspended in the atmospheric air; for instance, it has been repeatedly observed at post-mortem examinations that particles of cotton, lint, or wool, as well as coal dust, emery powder, and particles of steel, are inspired and incorporated in the lung substance, and several cases of chromate of lead poisoning have been recorded in which inhalation of the dust occurred while weaving yarn colored with this pigment. These cases will be referred to under the head of that poison.

The atomization of liquids, now so common a means of medication by inhalation, furnishes a very efficient avenue for the administration of drugs, though the chances of its use in criminal poisoning would not be probable.

The uterus furnishes also a very rapid means of absorption (as, for instance, in cases of carbolic-acid poisoning); and often, from carelessness or ignorance on the part of the practitioner, substances apparently nonpoisonous may, by local contact with the outlet of the Fallopian tubes, originate an irritation which may finally effect a more extensive and even fatal salpingitis or peritonitis. There have been reported in medical literature cases where an attendant, not having been cautioned in advance, has injected, *per vaginam,* water mixed with air from a bulb syringe into the uterus after parturition, thus making a break in the column of water in the injection tube; then water mixed with air passed into the flaccid uterus. The presence of the warm injected fluid caused the muscular wall of the uterus suddenly to relax and contract alternately; in this way air passed into the large veins, and so on into the heart and stopped its pulsations. The consequence of such an accident will inevitably cause a sudden death.

Intravenous injection of chloroform, in the writer's experience, has also produced the effect of introducing globules of chloroform into the column of blood on its way to the heart through the large veins, and so stopped the heart's contractions.

When the serous membrane is irritated or inflamed it will the more readily absorb a drug or poison; and thus a smaller quantity than under normal conditions may be followed by serious and, possibly, dangerous results.

It is undoubted that certain substances, innocuous in themselves when introduced in the ordinary channels, may, when introduced into extraordinary channels, provoke an irritation which can cause, and has secondarily caused, fatal or serious inflammatory processes; for instance, fistulous openings into the chest, or abdomen, or by the urethra into the bladder, etc. There are certain substances which may be introduced into the mouth and stomach, and are there so slowly absorbed and rapidly eliminated at one and the same time that poisoning may not occur, while, if the same drugs be directly introduced into the circulation, a very small amount acts as an active and often fatal poison; this is also true of similar substances introduced through the cellular tissue; as, for instance, by subcutaneous injection or by a wound. The interior surfaces of the mouth, nose,

and pharynx absorb actively many poisonous substances, even if they are not swallowed, and hence give rise to fatal poisoning; among these substances may be mentioned cyanid of potassium and, especially, nicotin.

The inner folds of the prepuce contain a good absorbing surface. The inner surface of the bladder does not usually offer a good avenue to absorption. The reason for this is obvious; the inner surface of the mucous membrane of the bladder is, under normal conditions, an envelope in which the tissue does not absorb the liquid contents of the bladder. It is ordinarily a receptacle to store an excretion, and when its walls are distended, the muscular fibers react and the bladder then contracts to expel the liquid contents; yet we know that when some drugs, such as belladonna, are present in the system, their action produces a relaxation and consequent elongation of the muscular (especially those unstriped or so-called involuntary) fibers. In this condition the elimination or excretion of the drug may be delayed, and be held in the tissue circulation. In this case repeated doses may cause the accumulation of an amount which may produce a serious and dangerous effect.

The alkaloid derivative from belladonna called atropin may be absorbed from the bladder even when it has been eliminated from the system by the kidneys, and hence arises the necessity for catheterization in the treatment of atropin poisoning. But a very important channel for the introduction of drugs and poisons in a state of solution is that of the cellular tissue. From the peculiar character of this tissue, medicines, especially those of a nonirritating kind, are readily introduced by means of a hollow needle, through which the fluid is forced by pressure from a small syringe into the cellular or subcutaneous tissue, and very rapidly (from five to ten minutes) peculiar physiological or medicinal effects are produced. Morphin presents a fair illustration of this fact, because a half or a third of the dose which would be required by the stomach will, in ten or twenty minutes, exhibit the full action of the drug if administered by this means. Ipecacuanha, whose emetic properties are well known to follow the swallowing of a proper dose, will exhibit emetic properties, though not so violent, when injected under the skin. Apomorphin, an artificial drug, will produce more violent emesis when given subcutaneously than when swallowed. This fact is now so well known to the medical profession that it is hardly worth while to discuss it in further detail here.

The skin in a healthy state absorbs but few medicines, and these

only in small quantities unless in a finely divided state as when combined with some fat, as in the use of ointments. Mercury and arsenic offer a good illustration of this method of drug administration by inunction. There are, however, some substances which are absorbed by the skin with considerable energy, as, for instance, an external remedy for skin diseases called chrysophanic acid. Gaseous or the more volatile substances are absorbable by the skin, while substances simply soluble in certain solvents do not readily pass through the cutaneous surface, and when dissolved in water, the natural excreting action of the skin tends to exhalation rather than inhalation, and thus offers a difficulty to the absorption of the dissolved substance; though the palms of the hands and the soles of the feet, as well also as the armpits and groins, present a slight exception to this statement. On the other hand, under certain conditions of heat and bodily exercise, certain substances in contact with a large surface of the body are absorbed quite actively. There are recorded cases of soldiers,[16] who, having secreted next to their skin tobacco leaves, and after walking sufficiently to excite an active perspiration, have been overwhelmed by the absorption of nicotin, and thus been fatally poisoned; there are also instances where warm cataplasms or poultices of tobacco applied to the skin of children by ignorant and careless persons have caused fatal poisoning by nicotin; poisoning has also been caused by the application of belladonna plaster to the skin.

Moreover, it should not be forgotten that, where there is a considerable abrasion or loss of cuticle, the absorption of many substances in solution can readily be effected, as, by the endermic method of administering drugs; absorption of many substances may also be facilitated by a diseased condition of the skin, as, for instance, in eczema and in skin diseases peculiar to syphilis, many medicinal substances are readily absorbed which a healthy, unbroken skin will not absorb. The serous surfaces in chest or abdomen, when inflamed, also offer a very ready means of absorption.

Dr. Arthur P. Luff,[17] in discussing the symptoms of chronic mercurial poisoning, calls attention to some cases where the application of a "so-called skin tonic to the skin, a solution of some mercurial salt, for the purpose of improving the complexion, produced somewhat severe mercurial poisoning; and the case in question came be-

[16] Gallavardan, Empoisonnement par l'Application des Feuilles de Tabac sur la Peau, Gazette des Hôpitaux, 20 Août, 1864, from Tardieu, Op. cit. 1875, p. 929.

[17] Text-book of Forensic Medicine and Toxicology, Vol. I. pp. 191, 192.

fore the Dublin law courts." He mentions another case where acute periostitis of the jaw followed the external use of mercurial lotion.[18] In regard to the absorption of arsenic into the general system after its external application to the skin, we quote from the same work: "Death has occurred on several occasions from the external application of arsenic; in such cases arsenic is found in the viscera, including the stomach; the arsenic is carried to the different viscera dissolved in the blood, and may pass into the contents of the stomach by a process of transudation or osmosis. In a case in which an ointment containing arsenic and white precipitate (mercurial salt) was applied to the diseased head of a child, death occurred after ten days, and at the post-mortem examination the mucous membrane of the stomach and intestines was found red and inflamed, and traces of arsenic were discovered in the fluid contents of the stomach, and in the liver. This transference of absorbed arsenic from the blood to the stomach and intestines has been distinctly proved by the experiments of Taylor and Pavy.[19] Plasters containing arsenic have been applied by quacks to cancerous and other tumors, and have produced death, as a result of the absorption of the arsenic to the diseased surface. In 1878, a number of children in Essex were killed from the employment of a dusting powder, supposed to be violet powder, which was dusted onto their skins after they had been washed. This powder was subsequently found to contain one third of its weight of arsenic. From one of the bodies Tidy extracted 6½ grains of arsenic."

These various illustrations of the absorption of poisons from the external application of them to the healthy and diseased surfaces of the skin show that this is an important subject for consideration.

9. What constitutes a case of poisoning.— We come next to the consideration of what constitutes a case of poisoning. As facts in toxicology are becoming better known, it is somewhat difficult to make a general statement which would lead to suspicion of the administration of poison. In general terms it may be said, in cases where there are no marks of personal violence upon the body of the deceased, and the history of the symptoms can hardly, in the opinion of a competent medical practitioner, explain a sudden and suspicious death as attributable to natural causes, there is reason to suppose that the deceased came to his death either from some internal arrest of the vital functions by a naturally explained accident, whose pathological effects can be shown at the autopsy, or else by means of some poison which

[18] Op. cit. and Br. Med. Jour. 1893. [19] Guy's Hospital Reports, 1860.

has been absorbed. In the more evident cases of poisoning, for instance, by an irritant or corrosive agent, or by a poisonous metallic agent, or poisonous alkaloid, etc., the history may be generally comprised in the following words:

Whenever a man in apparently full health is suddenly overtaken with serious and increasingly alarming symptoms, accompanied with pains in the region of the stomach; or when, in the absence of vomiting and diarrhea, a complete prostration of the vital forces, a cadaverous expression of countenance, and an abundant perspiration suddenly appear, and are soon followed by death, there is good reason to suspect poisoning, the attestation of which must be proved by anatomical and chemical examination of the body. In certain states, that of Massachusetts chiefly, the statutes which provide for suspicious deaths require that the official medical examiner, duly commissioned as such by the executive, shall proceed immediately to view the body, and examine the circumstances of death. This official, skilled in the science of medicine, unless satisfied that the death is due to natural causes, is required to take charge of the body, and to satisfy himself, not a jury, of the cause of death. This disposition of the investigation combines all the advantages of the Scotch method and that of the French system, because it allows a medical man to use his medical education and judgment in forming a professional opinion, which must immediately be reported in writing to the legal authority—judge and district attorney—which is charged with the inquest as to who caused the death. If the district attorney is for any reason dissatisfied with the reported professional opinion of the cause of death, he may order an autopsy to be conducted. According to this process, which, in our opinion, is superior to the coroner system, any sudden death may be a supposed case of violence, and upon this supposition the medical examiner is required to investigate and report upon the physical cause of death.

The object of the medical expert here, as in all other attempts upon the health or life of an individual, is to determine in a precise manner the nature of the disease or cause of death; but, in the majority of crimes, the expert who is called upon to investigate these causes may not begin his examination for weeks or months after the commission of the deed, and until suspicions have led to judicial interference; hence, the natural difficulties the expert would experience are greatly enhanced by the unnatural delay.

10. Unreliability of symptoms.— The great difficulties connected with the discovery or determination of the nature of the symptoms

which have preceded the death are hampered by the character of the source of information; *viz.*, the unwillingness and ignorance of those who witnessed the symptoms; and the fact that a great number of poisons cause symptoms that are almost identical in character, and similar to those of many diseases.

On the other hand, the symptoms following the absorption of some of the poisons are very similar, and it may be extremely difficult for an expert to determine by the history preceding death that a certain drug had been administered. It is hardly necessary to point out in this place in what way he may be misled or confused, as under the head of each poison the symptoms will be carefully given, and it will be seen that these alone can hardly be relied on to settle this question definitely.

Here the attention of the reader should be especially called to a very important consideration in connection with our discussion of the part which a description of a train of symptoms bears to the suspected death by a poisonous agent. In the course of the trial of Mrs. Robinson[20] for murder, before the Middlesex session of the Massachusetts supreme judicial court on February 6, 1888, the physician who attended the victim during her last illness, admitted on cross-examination that he filed a certificate of the cause of death as the result of pneumonia. The five suspicious deaths which were closely associated with the presence of Mrs. Robinson in the house in each case, led to exhumation of the dead bodies, and a judicial inquiry as to the cause of death in each one. The result of the trial was conviction of the accused of the crime of murder of one of the victims by administering arsenical poison, and sentence of death was pronounced by the court on June 28th of the same year. In one of these cases the evidence showed that, during the early portion of the physician's attendance upon the victim, and before the arrival on the scene of the murderess, she was certainly ill with pneumonia, and convalescence had begun before Mrs. Robinson came to stay in the house. The physician testified that then a new set of symptoms appeared. These new symptoms were not in accordance with symptoms of pneumonia. Notwithstanding this fact he filed his opinion that her death was the result of the disease pneumonia. When, however, the chemical expert gave evidence in court that there was proof of the presence of a large amount of arsenic in the cadaver after its exhumation, he

[20] The official report of the trial of Sarah Jane Robinson for the murder of Prince Arthur Freeman, in the supreme judicial court of Massachusetts, published by the attorney general, under chap. 214 of Acts of 1886; Boston: Wright & Potter Printing Co. State Printers, 1888, pp. 112–115. See also Case VII. in Appendix.

changed his opinion as to the cause of death, which he then attributed to poison by arsenic. All of the above evidence goes to show how little bearing symptoms, unaccompanied with the proof of the presence of a poison within the tissues of the cadaver, will have in the formation of an opinion which will stand cross-examination. Another point is brought out in this trial, and it is of sufficient importance to be mentioned in this preliminary chapter: Every physician who is called to attend upon a patient showing symptoms which apparently have occurred most suddenly and appear serious enough to endanger life, must have in mind the possibility of the ingestion of poison as a cause of the symptoms. The history of the evidence, not only in the above case, but in many books on toxicology, shows that the neglect of such caution has often caused great difficulty in establishing, upon probable and certain grounds of truth, a case in doubt when afterwards brought to trial before the court of adjudication. A little forethought on the part of the attending physician might have led him to collect vomitus, clothing, and other articles upon which poison may have fallen, and thus by chemical analysis establish or remove a doubt.

11. Points to which attention of medical attendant should be directed.—Dr. Luff, in the text book previously referred to,[20a] mentions certain points to which the attention of the medical attendant should be chiefly directed, apart from the treatment of the patient:—

"1. The time at which the symptoms commenced, and the nature of the symptoms.

"2. The time at which the symptoms commenced after the last ingestion of food.

"3. The occurrence of any recent previous illness from which the patient may have suffered.

"4. If the patient has vomited, the vomit should be collected, or, if necessary, scraped up from the floor, or from the dress, bedding, or carpet; if necessary, a portion of the dress, bedding, or carpet containing the vomit, should be cut out and preserved.

"5. The nature of the food recently taken by the patient should be ascertained; and if suspicion attaches to any articles of food, these should be secured by the medical man and preserved under seal.

"If the death of the patient occur, in addition to attending to the points above mentioned, note should be taken of the following:—

"(1) The exact time at which death occurred.

[20a] Text-book of Forensic Medicine and Toxicology, Vol. I. p. 111.

"(2) The position of the body with regard to surrounding objects; its attitude; and the condition of the dress.

"(3) All surrounding objects should be carefully observed; and any bottles, packets, or weapons in the room, should be collected and preserved.

"(4) The condition of the body as to lividity or pallor should be noted, and also whether the countenance presents a distressed or calm appearance."

We should also remember that, if there is found near the dead body, or in the room or place where it lies, any vial, cloth, or article of furniture which may be supposed to point to any suspicion of the substance used, it should be carefully taken in charge by the investigating officer and examined. Oftentimes a careful search of the premises and person of the dead body will reveal some suspicious article which must not be overlooked. Briefly stated, the action of narcotics is so often similar in symptoms, that a guess of the specific poisonous agent is not admissible. The same remark may also apply to many of the alkaloids the symptoms of which are so rapidly followed by fatal issue that, unless a man skilled in medicine should himself have seen the symptoms preceding death, no reliable history of them can be obtained, since it is not safe to trust to the hearsay evidence of bystanders. The same may be said of certain natural accidents which cause sudden death, and which may erroneously be attributed to a poisonous agent, such, for instance, as aneurysm, embolism, or internal hemorrhage. Especially does the preliminary examination require tact on the part of the investigating officer.

The importance and characteristics of the medical expert are more particularly discussed in another volume of this work (see Vol. III., Chap. 27); yet it is well to call attention in this place to a very important caution which every expert should keep constantly before him. He is assumed to be specially qualified, both by his education and actual experience in his professional practice, to be able to describe in a clear and precise manner the evidences of death by natural causes, and the pathological appearances which are the results of these causes, whether they are the consequence of natural disease, natural accidents, or from violence at the hands of the deceased or at the hands of another. In presenting his testimony before the court of inquiry he should confine his statements to facts within his own knowledge and experience, and not indulge in theory. "It is chiefly in the endeavor to obtain evidence upon intricate and novel questions that the expert stands in danger of traveling too far beyond into the

domain of theory, into which he is led as far as he can be made to go by the counsel who employs him."[21] He is required to defend his opinion which he may present as his testimony before the court, and he should do this in a way that would carry conviction to the mind of an ordinary juryman to show that his opinion is safely founded upon his medical knowledge and personal experience. He should, as much as possible, avoid the use of too technical and medical phrases: these phrases would not convey to an intelligent juryman the evidences of abstract appearances, though to the medical expert himself they clearly represent these same appearances. He should also, in order to present intelligently to an audience not familiar with the physical appearances which he himself has seen, describe, from notes which he should have taken upon his first view, when officially called, of all articles about the dead body and their relation to it, the appearance of the room, the furniture in it, or the outside surroundings (if the body is not in a building), and whether the surrounding locality has the appearance of having been disturbed by human struggles or violence.

It is very important to interrogate the bystanders, not by leading questions which would suggest the direct answer which is in the mind of the questioner, but simply to collect immediate information as to facts which have been observed by them, before they forget the observed facts, or have their memory embarrassed by the opinion of others. These notes, taken at the earliest moment during the "view," may be of great value in supporting a theory that death resulted from some accident or incident which preceded the death. Sometimes a heap of offal or the presence of filth may have an important bearing upon the inquiry as to whether food taken by the deceased may not have been mixed with some organic vegetable or animal decomposed matter; as, for instance, a ptomain poison; there may be some sausage or piece of decayed meat lying about the quarters which should be taken in charge for future examination.

12. Absorption of poison.—We have, in the preceding pages, referred to the absorption of poisons into the human body. No matter how the poisons may be administered to any person, if we except the local action upon the external human tissues, they act by absorption into the systemic circulation of the blood, and then destroy the living functions of the cells, for which they have an affinity. Provided the functions of eliminating organs retain their properties for excreting

[21] Witthaus and Becker, op. cit. p. 98.

the poison, a portion of the poison may pass out of the system with the excretory fluids.

Poisons act by absorption[22] (though certain of them may cause local lesions by their irritant properties), and are carried by the blood through all the tissues. They are eliminated with the excretory fluids. On the ratio of elimination to absorption depends the activity of these poisons. If a drug is absorbed more rapidly than it is eliminated, we get symptoms of the action of this drug. Unless the inhalation of ether exceeds its exhalation, no etherization is produced. Unless prussic acid is absorbed more rapidly than it is eliminated, no poisonous symptom follows. In certain animals the elimination of a drug is so rapid that it is extremely difficult to cause death by the administration of this drug. Belladonna is eliminated very rapidly by goats, and it is almost impossible to destroy their life by this drug. It is known, for example, as a fact reported by the writer in his graduation thesis,[23] that it is impossible to kill a horse by the introduction into his stomach of a fluidounce of the officinal prussic acid solution of the United States Pharmacopœia; an amount far in excess of what would kill many of the other warm-blooded animals, a teaspoonful being sufficient to destroy man. It is quite within the range of probability that this exemption of a horse from what is generally admitted by most authorities on toxicology to be a lethal dose for an adult man can be explained by the supposition that this drug is so rapidly eliminated from the blood that not enough of the poison is there retained to cause the specific poisonous action through the reflex nervous system centering in the medulla oblongata upon the lungs and heart. It will be seen by reference to the thesis referred to that the introduction of the poison was not only by ingestion, but also by subcutaneous injection, and that it required frequent and very large

[22] On the trial of Elizabeth McCraney at Otsego, New York, in December, 1860, for the poisoning of Huldah McCraney, the evidence was that the deceased, during her illness, exhibited, in the opinion of several experts, symptoms common to arsenical poisoning, and that arsenic was found in the remains. The case of the prosecution was that the arsenic had been administered by injection into the rectum. There was some conflict among the experts in their opinion as to the cause of death, and the defendant was acquitted. (*People* v. *McCraney*, 6 Harris C. R. 49, where the medical testimony is reported in full.) That poison may be administered by injection, and be detected by the tests heretofore announced, is well established. (*Ibid.;* Taylor on Poisons, 2d Am. ed. pp. 111, 229, 373; Taylor's Med. Jur. 4th Am. ed. 68; Beck's Med. Jur. 11th ed. 447; London Lancet, Dec. 22, 1855; Medical Times and Gazette, Dec. 22, 1855; Edin. Med. Jour. Jan. and Feb. 1856, cited in 6 Parker's C. R. 110.)

[23] Thesis on The Poisonous Action of Hydrocyanic Acid on Animals, published by authority of the Medical Faculty of the Harvard Medical School, in the Boston Med. and Surg. Jour. Aug. 23, 1866.

doses hypodermically injected, before the horse succumbed to the action of the drug, and that the usual phenomena noticed in other animals preceded his death. It is not so much the quantity of a special drug administered by the mouth or other channels of introduction, as it is the amount which is absorbed by the particular tissue whose function it destroys.

The recent work of Prof. Rudolf Kobert on Practical Toxicology[24] explains the action of poisons, when absorbed locally and remotely, so tersely and in accordance with more recent knowledge, that we do not hesitate to present his views in the following quotation:

"Some of the agents, such as the salts of the heavy metals, will readily combine with the protein substance, thereby causing their destruction, *viz.*, necrosis of the tissues involved; others, such as concentrated acids and caustic alkalies, act also as powerful irritants and cause a reactive inflammation; still others, as strychnin, morphin, curare, and muscarin, cause an excitation or enfeebling of the nerves, muscles, or glands of the affected parts, without any marked apparent changes.

"The remote effect is produced by the absorption of the poison into the lymphatics and into the blood, causing general symptoms and diseases of other organs, *e. g.*, of the kidneys, following the administration of cantharidin; of the brain, after taking opium; of the intestine, after quillaic acid. Practically, the remote action is really a local one, produced by the poisoned blood circulating everywhere.

"The poison, as it circulates in the blood, may be either decomposed, or it may enter into the combination with the blood constituents and thus change the composition of the blood, or it may reach the various organs in its original condition. Physiology teaches us that various endosmotic changes take place in these organs, depending upon their functions, upon the formation of their constituent elements, and upon the number and arrangement of the capillaries passing through them. The chemical constitution and physical properties of the poison will determine, to a varying degree, the role it plays in these changes upon participating in the interaction of the vessels of the tissues. The presence of this foreign substance sooner or later disturbs, to a greater or less degree, the healthy condition and function of the organs particularly affected; and, again, this cannot take place without a reaction upon the whole body. The animal or-

[24] Translated by L. H. Friedberg, Ph. D. Am. ed. N. Y. Wm. R. Jenkins. 1897, p. 8.

ganism, however, possesses four means of rendering partly or entirely harmless, poisons which have entered the system:

"1. Rapid elimination. Under this head, naturally, we first mention vomiting, which, fortunately, occurs so promptly following the introduction of most poisons into the stomach that it generally saves the life of a patient, or at least has already materially lessened the danger to life before the physician puts in an appearance. We should call this vomiting, which takes place before the absorption of the poison, primary vomiting, in contradistinction to a secondary emesis, which takes place following absorption, and which latter is either exclusively a sign of disturbed cerebral activity, or is caused by the excretion of the poison from the blood into the stomach. In an analogous manner we must differentiate between a primary diarrhea, which carries off the poison before absorption, and a secondary purging, which is a sign of disturbed intestinal innervation, or is caused by the excretion of the poison from the blood into the lumen of the gut. Some poisons are not removed by vomiting or by purging, but appear in the urine within a remarkably short time. Thus, for example, it is impossible to produce complete curarization by the administration of moderate, though oft-repeated, doses of curare, because the excretion of the poison through the kidneys takes place as rapidly as does absorption. The liver, pancreas, gastric mucous membrane (for morphin), intestinal mucous membrane (for mercury), salivary glands, mammary glands, and lungs, are other channels effective in assisting the excretion of various substances from the blood. Not nearly enough attention was formerly given to the excretion through the glands of the mucous membrane of the stomach. Finally, elimination takes place through the structures of the skin, especially through the sweat glands.

"2. The organism deposits and fixes poisons, in a manner not yet sufficiently understood, in several organs, especially in the liver, which certainly must be regarded as a filter for poisons, so far, at least, as enzymes (*e. g.*, emulsin), metals (iron), metalloids (*e. g.*, arsenic), and alkaloids (*e. g.*, strychnin), are concerned. It is probable that, in the case of some substances, the biliary acids play an important part in the matter. We can hardly imagine that this disposition is accomplished in any other way than in the transformation of the readily soluble poisons into saline combinations, not freely soluble (bile-acid-alkaloids), or into albumen derivatives (metalbuminates). But, since these combinations are in no case entirely soluble, the beneficial

action of the liver consists only in the fact that it gives the acute poisoning a more protracted, and consequently a milder, course.

"3. The organism renders the poisons innocuous by phagocytosis. This destructive crusade carried on in the interest of the body by phagocytes, which has not yet been sufficiently inquired into pharmacologically, is applicable for certain toxalbumins (toxopeptone, enzymes), as well as for heavy metals.

"4. The organism transforms the poison into a comparatively harmless, though readily soluble, combination. Such a transformation may consist of neutralization, oxidation, reduction, coupling, splitting, and peculiar changing of the chemical constitution. 1. As an example of poisons rendered inert by neutralization, we must mention the acids, which are transformed as far as possible by the organism into the corresponding alkaline salts of less poisonous, or absolutely nonpoisonous, properties. So far as the stomach is concerned, the organism attempts to balance any excess of alkali by the acids of the gastric juice, and does the same thing in the blood by the decompositions of an immense number of blood corpuscles, whereby glycerophosphoric acid is formed from lecithin. Caustic lime is combined with carbamic acid, and then excreted. 2. The best known example of inertia produced by oxidation is that of phosphorus, which is transformed into phosphates. In an analogous manner the extremely poisonous sulphids are converted into sulphates which are relatively nonpoisonous. The organic acids and their salts are oxidized to the ultimate degree, producing carbonates; and it is a prominent and important fact that in the latter case the dangerous diminution of the alkalescency by means of these acids, is transformed into an increase of alkalescency, since even the bicarbonates are of alkaline reaction. 3. Examples of producing inertia by means of reduction are offered in the cases of iodates, chlorates, and perchlorates, which are excreted in the markedly less poisonous form of chlorids and iodids. 4. Inertion produced by coupling is one of the most remarkable facts in physiological chemistry. An intimate knowledge of this phenomenon is as imperative for the physician at the bedside as for the chemist intrusted with the chemical analysis of the remains. A poison can unite by coupling: (*a*) with sulphuric acid (*e. g.*, phenol and cresol); (*b*) with glycuronic acid (*e. g.*, camphor, borneol, menthol); (*c*) with glycocoll (*e. g.*, benzoic acid, anisic acid, a part of salicylic acid). 5. Inertion produced by splitting occurs with tannic acid of nutgalls, and with some glucosids (*e. g.*, salicin). 6. Examples of

changes peculiar to themselves, as productive of inertia, are offered by the salts of ammonia, which are transformed into urea.

"The liver is the most important organ in producing changes in poisons peculiar to themselves. Coupling occurs partly in the liver and partly in the kidney. Splitting processes take place mainly in the intestinal canal, although the liver must also be considered in this connection.

"For a time it seemed that we were justified in supposing that organic substances could be divided into two well-defined classes, according to their respective actions exhibited within the animal body; the substances of the fatty series were supposed to be destroyed, while those of the aromatic series were not. To-day we know that this does not hold good for all substances; not even oxamid, belonging to the fatty series, a trace of which is oxidized; and tyrosin, a member of the aromatic series, which can be completely transformed into urea, carbon dioxid, and water.

"This observation, therefore, can at the present time be stated only in the following form: Organic substances containing annular linkage within the molecule are frequently not oxidized to form carbon dioxid, water, and urea. It is immaterial whether or not they belong to the aromatic series proper. Substances not containing annular linkage, which are oxidized with difficulty or not at all, are mainly certain amids.

"One of the foremost tasks of scientific pharmacology is to explain the relation between the chemical structure of a substance and its pharmacological action. Unfortunately, it can only be said at present that uniform laws, which would be of great service to the physician, have not yet been discovered. This is neither the time nor place to dilate upon the many interesting structures of such laws."

The kidneys, the large bowel (colon), and the lungs are especially active in eliminating, or, in other words, expelling, the poisonous substance out of the system. The liver assists materially by means of its active liver cells, which will select certain poisons[25] "such as iron, copper, lead, arsenic, etc., in its parenchyma; and the alkaloids, nicotin, quinine, morphin, cocain, atropin, hyoscyamin, strychnin, and veratrin, and curare, and ammonia are arrested in their absorption by the liver." The authors quoted also state that "the protecting influence of the liver is directly proportionate to the amount of glycogen present. This accumulation or deposition of poisons in the liver, which renders

[25] Witthaus and Becker, op. cit. Vol. IV. p. 82.

that organ the best situation in the cadaver for the detection of absorbed poison, can only be explained on the supposition of the conversion of the dissolved substance into a less soluble combination, which is deposited." Disease of the tissue cells in these eliminating organs has, therefore, an important bearing upon the question as to what relation the portion of a given amount administered in poisoning cases bears to the amount absorbed in the body in a case of suspected poisoning. If the eliminating organs are diseased to such an extent as to interrupt their function of expelling, during a fixed period of time, what, in health, would be a normal amount, a larger portion of the poison administered would be retained than should be expected in a person in whom the same organs are healthy, or in a condition of normal functional activity. The medical expert is therefore cautioned to examine carefully by the eye and through a miscroscope the cell tissue and the parenchyma of the kidneys, liver, and the colon (large bowel), for the purpose of ascertaining the normal or abnormal, the anatomical and also the physiological condition of the cells. This result will assist him to define in any case what, in his opinion, should be considered a specially lethal or dangerous dose, and to give his reason therefor. In other words, a dose or quantity of a drug, by the ingestion of an amount smaller than would be given if they were in a normal condition, may, in a diseased condition of these organs, be followed by dangerous symptoms.

Witthaus and Becker state that "it is quite possible that the poison may be quite removed by vomiting and elimination, yet death result from its action. In that event, clearly, the poison may be detectable in the urine or vomit, but not in the cadaver. Such a case is reported by Taylor in which a man died seventeen hours after taking about an ounce of arsenic." Attention should be called to the information furnished by more recent discoveries in chemistry, by means of which new methods for the detection of the presence of arsenic in the human body are practised in chemical analysis.[26] Prof. Charles R. Sanger has described a much more delicate process for the detection of arsenic in the urine than by the older method. Prof. E. S. Wood, of Harvard Medical School, presented a communication on the 17th day of November, 1891, on the "Contribution to the Study of the Elimination of Arsenic," by which he proved that the presence of this drug can be detected by Prof. Sanger's method, in the analysis of the urine from a person, who had been taking arsenic, during a period of ninety-

[26] Am. Acad. Arts and Sciences, Proceedings, Vol. XXVI.

three days from the time of the last and large toxic dose of arsenic.[27] Prof. Wood calls attention in this communication to this fact: "It is stated in all works on toxicology and therapeutics that, after medicinal or toxic doses of arsenic, the drug is completely eliminated in about three or four weeks. In support of this, numerous cases are cited, particularly (Taylor) the famous case of Dr. Alexander, who died sixteen days after taking, accidentally, a large dose of arsenic, and in whose organs, after death, no arsenic could be detected by chemical analysis by this eminent toxicologist."

Therefore, in view of the probability that the advanced methods of chemical analysis may trace poisons within the excreta of the human body by new and delicate methods not previously known to the ordinary medical practitioner, the medical attendant should take especial pains to preserve all the excreta from the eliminating organs, so that, if the judicial inquiry demand a very thorough search for a poison, these excreta should be at hand for an analysis. Like all the other parts of the body, these should be collected, marked, and sealed in the presence of witnesses.

Witthaus and Becker[27a] note that in two cases of suspected poisoning which they cite, the attending physicians omitted to preserve the food or drink prepared for their patients, as well as the excreta from the organs of the body; in consequence of their neglect an important element to substantiate evidence was lost. In one of these cases the undertaker had, previously to the examination required by the inquest, embalmed the body. In this case the suspected poison was arsenic, which is frequently used in the embalming fluid.

13. Accumulated action of certain drugs.— It is a fact well known to physicians that certain poisonous drugs produce an action upon the human system which is called "accumulated." Without giving an explanation of what this term means, as usually interpreted, it is sufficient for the purpose of toxicology simply to give, as an illustration, the so-called accumulated action of one of this class of drugs,—arsenic: this is sometimes observed in the ordinary treatment of disease by doses which are nonpoisonous in normal condition of the organs, that is, in health; the continuous ingestion of this drug will, in some, but not in all, cases provoke marked local irritation upon the digestive tract,—especially upon the mucous surfaces of the stomach and intestines; even when it does not produce this local action, a long-continued administration of this poison in nonlethal doses may induce a fatty

[27] Boston Med. and Surg. Jour. April 27, 1893.

[27a] Op. cit. Vol. IV. p. 82.

degeneration of the liver tissue; it may extend this action and cause fatty degeneration of the tissue of other organs. In consequence of the fatty degeneration of one or more of these organs, interference with their normal functions would naturally prove a menace to health. This is usually due to the fact that the drug is not completely eliminated by the excretory organs of the body, and, consequently, this accumulative action upon the tissue of these organs induces an abnormal condition. This condition may continue for some time after the administration of the drug has ceased. The same is true of other poisonous drugs, such as phosphorus, antimony, etc. Again, belladonna will, after prolonged and continuous use in moderate, small doses, be followed, even after its discontinued ingestion, by a prolonged and continued display of its peculiar physiological effects. This is due, not to any selective action upon the organs of the body, but to its special action upon the unstriped or involuntary muscles in the body; and, in the case of belladonna, we do not expect to find any fatty degeneration of the muscle tissue. The cause of the accumulated action of belladonna is the incomplete elimination of this drug by the excretory organs of the body. However, in the case of the class of drugs first mentioned, the cells of the organs which select and hold these in their tissue seem to form a combination with the drug, which alters their physical condition and produces a suspension of their function. When we, later on in this volume, discuss the pathology of each drug here mentioned, we shall treat of this action under the head of chronic poisoning, to which it more correctly belongs.

The fact must not be lost sight of that some poisons even in non-poisonous doses may, in certain conditions of disease or bodily weakness, cause untoward results (see § 4).

14. Mode of invasion of symptoms.— In most cases of acute poisoning, by which is meant those in which a single dose capable of destroying life is taken, the symptoms arise more or less suddenly. This, in a great measure, depends upon the rapidity of absorption, because the more rapidly a poison is absorbed the quicker will poisonous symptoms be manifested (see § 12). In the chapters upon the individual poisons, the length of time elapsing before the accession of the symptoms will be given; it varies with each poison, and is influenced also by several circumstances, such as the fullness or emptiness, or the healthy or diseased condition of the mucous membrane, of the stomach; the state of health and the habits of the individual, as has been already stated. While the symptoms of poisoning arise suddenly after eating or drinking, these symptoms are not necessarily a proof

of such poisoning, and the medical attendant must be careful not to suspect these symptoms as due to the ingestion of a poison, merely from the suddenness of their appearance. We have specially mentioned that a suspicion should arise when the patient in previous health is attacked with violent symptoms, such as vomiting, pain at the epigastrium etc.; but this may occur from a sudden attack of illness, especially at a time of a cholera or yellow fever epidemic, or from indigestion during a period of intense summer heat. Although arising suddenly, the symptoms do not necessarily follow immediately the ingestion of the poison. If it has been swallowed in food or drink, the symptoms announcing the fact of poisoning may not come on for an hour or more afterwards. This fact has been frequently observed in arsenical poisoning, and is usual in poisoning by opium, belladonna, digitalis, and some other narcotics. But when the symptoms have begun to manifest themselves, there is a progressive development of them, and they present (like any disease) certain features, which, combined, form a portrait by which they may be referred to some one class of the poisons, or be known to depend upon a particular poison. It is not, indeed, meant that there may not be a remission of the symptoms in poisoning, due either to the influence of treatment or to the spontaneous struggles of nature; but this circumstance, which is more apt to take place when a dose insufficient for the destruction of life has been taken, can hardly affect the value of the sudden accession and development of characteristic symptoms. It should not be overlooked that poisons have sometimes been taken or administered otherwise than by the mouth, as has been already stated in a previous section.

15. Duration of symptoms.—The duration of the symptoms is another consideration which has important bearings. Although sudden death is not produced by the majority of poisons, or at least by such as are usually swallowed, yet death from acute poisoning is an early result. A few minutes or hours may suffice; and, on the other hand, the patient may survive for days. No general rule upon this point can be laid down; arsenic usually destroys life within twenty-four hours, strychnin within an hour, opium within twelve hours, and prussic acid in a few minutes. Exceptions are seen to all of these general rules; and with no poison is there so wide a range in the duration of the symptoms as with arsenic, since it has been known to prove fatal in less than two hours, and after several days. The reader will find sufficient details upon this point hereafter.

16. Distinction between symptoms of poisoning and those of disease.—All the importance of the evidence derived from the symptoms depends upon the possibility of showing a distinction between them and a disease suddenly developed. This distinction should be sufficient, not merely to satisfy the mind of the physician, but to afford convincing proof to the jury upon the subsequent trial. This is often the most difficult and annoying duty of the physician; for while his own mind may be perfectly satisfied of the correctness of his judgment, he can rarely, with perfect conscientiousness, assert that the symptoms might not be explained upon the supposition of disease.

To illustrate: Dr. Vivian Poore, from whom we have quoted previously, gives an instance in a noted trial in England, namely, *The King* v. *Donellan*. The distinguished John Hunter was a witness in this case, where Captain Donellan was on trial for poisoning his brother-in-law, Sir T. Boughton, who swallowed a draught which the apothecary who furnished the medicine had stated contained no oil of bitter almonds. John Hunter attested (1) that the post-mortem signs were all due to putrefaction; (2) that death might have been due to apoplexy, and that, the head not having been opened, it was impossible to say whether this was true or not. Being asked on cross-examination whether the fact of a man in perfect health dying convulsed immediately after swallowing a draught did not point to poison, he replied, "If I knew the draught was poison I should say, most probably, that the symptoms arose from that; but when I do not know that the draught was poison, when I consider that a number of other things might occasion his death, I cannot positively answer to it." Hunter admitted that it was very probable that Boughton died from apoplexy. The final question put was:

Court: "Give me your opinion in the best way you can, one way or the other, whether, upon the whole of the symptoms described, the death proceeded from that medicine or any other cause."

"I do not mean to equivocate; but when I tell the sentiments of my own mind, what I feel at the time, I can give nothing decisive."

The judge (Hon. Francis Buller) made the following comments on the testimony of Mr. Hunter: "For the prisoner you have had one gentleman called; he is likewise of the faculty and a very able man. I can hardly say what his opinion is; for he does not seem to have formed any opinion at all of the matter. He, at first, said he could not form an opinion whether the death was or was not occasioned by the poison, because he could conceive that it might be ascribed to other causes. I wished very much to have got a direct answer from Mr.

Hunter, if I could, what upon the whole was the result of his attention and application to the subject, and what was his present opinion, but he says he could say nothing decisive. So that, upon this point, if you are to determine upon the evidence of the gentlemen who are skilled in the faculty only, you have the very positive opinion of four or five gentlemen of the faculty that the deceased died of poison. On the other side, you have what I really cannot call myself more than the doubt of another; for it is agreed by Mr. Hunter that the laurel water would produce the symptoms which are described. He says an apoplexy or an epilepsy would produce the same symptoms; but as to an apoplexy, it is not likely to attack so young and so thin a man as Sir Theodosius was; and as to an epilepsy, the other witnesses tell you they do not think the symptoms which have been spoken of do show that Sir Theodosius had any epilepsy at the time." The jury retired for an hour, about, and then brought in a verdict of guilty, and Captain Donellan was executed a few days thereafter.

Hence, this portion of the medical evidence cannot stand alone, but must be supported either by the positive correspondence with it of the post-mortem appearances and chemical analysis, or by the absence of any evidence from the autopsy confirming the notion of disease; and, moreover, it will not answer to place reliance chiefly upon any prominent symptom, but all of the symptoms must be considered as a whole. Thus, in poisoning by strychnin, the tetanic convulsions caused by this alkaloid might be plausibly ascribed to some other cause, if the convulsions alone were taken into account without reference to their number, the rapidity of their appearance, the condition of the intellect during the interval between the paroxysms, the duration of the case, or any of the other symptoms which contribute to make up the picture of a case of strychnin poisoning. Yet in the case of many poisons it is impossible from the symptoms alone to base evidence sufficiently strong to procure a conviction. Thus, should a person die with the symptoms of irritant poisoning, the physician would find it difficult to defend the position that similar phenomena might not be witnessed in an attack of cholera, or in gastroenteritis arising from some other cause. Dr. Lee says: "During the prevalence of malignant cholera in 1832, we mistook a case of poisoning by arsenic for an attack of this disease. A lady took more than a dram of the arsenite of potash, as we afterwards ascertained, with the intention of destroying her life, which was followed by severe retching, vomiting, cramps, a livid, cold, and clammy skin, and the other

symptoms which usually attend a severe attack of cholera."[28] Again, a recent writer on toxicology states:[29] "However striking and well defined the picture of strychnin tetanus may be, mistakes in diagnosis are rather frequent, especially when a medical man is hastily summoned, has never seen a similar case of poisoning, and has no suspicion of the possible nature of the seizure. If a young woman, for instance, is the subject, he may put it down to hysteria; and certainly hysteria not unfrequently affects similar convulsions. In a painful case in which the author was engaged, a young woman took or was given (for the mystery was never fully cleared up) a fatal dose of strychnin, and, though the symptoms were well marked, the medical attendant was so possessed with the view that the case was due to hysteria that, even after making the post-mortem examination, and finding no adequate lesion, he theorized as to some fatal hysteric spasm of the glottis, while there was ample chemical evidence of strychnin, and a weighable quantity of the alkaloid was actually separated from the contents of the stomach. Again, the medical attendant of Matilda Clover, one of Neil's victims, certified that the girl died from delirium tremens and syncope, although the symptoms were typically those produced by strychnin. Such cases are particularly sad, for we now know that, with judicious treatment, a rather large dose of this drug may be recovered from. If the case is a male, a confusion with epilepsy is possible though hardly to be explained or excused; while in both sexes idiopathic tetanus is so extremely similar as to give rise to the idea that all cases of idiopathic tetanus are produced by poison, perhaps secreted by the body itself. As for the distinction between idiopathic and strychnin tetanus it is usually laid down (1) that the intervals in the former are characterized by no relaxation of the muscles, but that they continue contracted and hard; and (2) that there is a notable rise of temperature in disease tetanus proper, and not in strychnin tetanus. Both statements are misleading, and the latter is not true; for, in strychnin poisoning, the relaxation is not constant, and very high temperatures in animals have been observed."

Dr. Arthur P. Luff, a high authority on toxicology,[30] states that "the convulsions of strychnin poisoning have been mistaken for the convulsions produced by certain diseases. The convulsions of tetanus are

[28] Copeland's Dict. Am. ed. article *Poisons*.

[29] Poisons: Their Effects and Detection. Alexander Winter Blyth; 3d ed. London: Charles Griffin & Co. Limited, Exeter Street, Strand, 1895, p. 331.

[30] Text-book of Forensic Medicine and Toxicology: London: Longmans, Green, & Co. 1895, Vol. I. 276.

those which are most likely to be confounded with the convulsions of strychnin poisoning, but they can be very easily distinguished. Of the varieties of tetanus, idiopathic and traumatic, the idiopathic is extremely rare in temperate climates, whilst in connection with traumatic tetanus a history of a previous injury may generally be obtained, such as a lacerated, punctured, or contused wound; although it should be borne in mind that a very trifling wound, such as may be inflicted by a splinter or by a rusty nail, may, after a lapse of several days, give rise to tetanus.

"The symptoms of tetanus usually commence from five to fourteen days after the infliction of the wound, but they may set in earlier or later.

"The convulsions produced by strychnin have been mistaken occasionally for those of epilepsy, but there should be no difficulty whatever in distinguishing between them. The mode of seizure, the loss of consciousness, and the peculiar clonic movements of the epileptic fit, are entirely different from the convulsions of strychnin poisoning. In addition, the deep stupor occurring at the end of an epileptic attack presents a marked contrast to the complete muscular relaxation, and the clearness of intellect between each strychnin spasm."

The danger of relying upon the symptoms alone, unsupported by the further and more certain proofs determined by the chemical expert, is sufficiently emphasized by the authors from whom the above quotations have been given as not to require amplified arguments.

It is evident that the symptoms observed after poisoning in some cases, as those, for instance, occurring in connection with the action of corrosive or irritant drugs, are caused by primary pathological lesions. Now these lesions may not immediately, that is, within two or three days or even months, result fatally; but their effects, which may cause destruction of tissue, perhaps of some important organ, as the stomach, air passages, lungs, etc., may give rise to secondary symptoms of disease, which are the direct consequence of the action of the drug. In a similar way, as stated in previous pages, account should be made of the slower effects of poisons which cause a deterioration of vital organs, such, for instance, as the fatty degeneration of the tissue of the liver, kidneys, or other viscera by phosphorus, arsenic, and other substances. These alterations of healthy tissue in a vital organ may slowly accomplish the death of the victim, and, without the history of the early symptoms, the ends of justice may be defeated. In these latter cases especially, the post-mortem examination may reveal a pathological condition which is not unfrequently ob-

served in disease; but the extensive distribution of the diseased tissue in the case of chronic poisoning, when it is not usually confined to one or two organs, may awaken a suspicion which the experienced pathologist will recognize as unusual in death from natural causes. In such instances an experienced pathologist would lend material assistance in expert testimony; and it must here be observed that the ordinary practitioner of medicine is not by any means an expert pathologist. The knowledge of pathological anatomy requires a large personal experience in autopsies, and microscopical examination of healthy and diseased tissues, as well as a careful educational training to interpret the results of his observation. It may also be further observed that the chemist may be an experienced pathologist and physiologist; and it should be the aim of the judicial investigation, as far as possible, to combine in one person the qualities of a trained pathologist as above stated, though it is evident that this cannot always be attained.

17. Post-mortem examination of a cadaver, in general.—This is frequently so important a question that preliminary precautions are essential to prevent mistakes which, later on, in the judicial inquiry, may cause confusion which cannot be rectified without danger to the truth of the inquiry; we, therefore, make no apology for entering upon a discussion of this part of our duty in our opening chapter.

The pathological condition of the viscera of the human body offered as evidence in court is not alone sufficient to establish a conviction, and a nice discrimination must be made between the effects of disease and those of a poison. The condition of these viscera can only establish a presumption, especially if the autopsy is not made soon after death. And every precaution must be taken in making the autopsy to eliminate any natural cause of death: every organ should be carefully examined, and every abnormal appearance should be carefully noted. Every circumstance, however insignificant it may appear to be at the time, must be noted. The external appearance of the body, whether livid or pale; the expression of the face, whether or not there are any marks of violence or injury, such as contusions or abrasions; the amount of post-mortem rigidity; and the exact time after death that the examination is made,—must be observed. Not only the appearance of the body, but its exact position in relation to other objects in the room, the temperature of the room, and all other circumstances and conditions, must be remembered by the physician making the examination; and it is best for him not to trust to his memory, but to

make a note of everything in writing, together with a plan of the surroundings of the body in any case of suspected poisoning.

Important information can frequently be gained by careful observation of the external appearances of the body and its surroundings; thus the existence of stains upon the skin and mucous membranes about the mouth or upon the clothing, suggests a corrosive poison, and the color of such stains may show the nature of the corrosive; the odor of certain poisons can sometimes be perceived about the mouth and nostrils, as in the case of prussic acid and phosphorus; the mouth and nostrils should also be examined for phosphorescence, which can sometimes be perceived in phosphorus poisoning; the condition of the pupil should be noticed, as in poisoning by atropin it will be found dilated; the color of the post-mortem discolorations (suggillations), which are due to blood, will be affected by some poisons, as, for instance, the bright rose-red in carbon monoxid poisoning, more rarely in prussic acid and cyanid of potassium poisoning, and the dark color in poisoning by the corrosive alkalies; the skin and conjunctivæ sometimes have a yellow color in phosphorus poisoning, and there may be found extensive ecchymoses. If putrefaction has commenced, its exact time should be noticed, since some poisons are apt to hasten, while others retard, it; as, for instance, in phosphorus poisoning putrefaction is liable to begin early, while in arsenic poisoning it is usually delayed, often for a very long time. The time at which the post-mortem rigidity appears may also prove an important question, since in some cases, as in strychnin poisoning, it is much hastened and lasts for a much longer time than usual.

In making the autopsy in a case of suspected poisoning, or wherever there is any liability of a legal investigation, special precautions must be adopted in order to guard against the possibility of a mistaken diagnosis. Everything done and seen should be noted at once. Every organ as it is removed from the body should be thoroughly and carefully examined, and, whenever necessary, a portion laid aside for later microscopical examination; for instance, for the detection of the fatty degeneration of the heart, liver, and kidneys so frequently caused by the action of arsenic, phosphorus, and other substances. All of the organs, including the brain and spinal cord, should be examined, so as to be sure that death was not due to natural causes. If any urine remains in the bladder, it should be removed and saved for chemical examination. After the organs have been removed from the body, care should be exercised in handling them; for instance, they should not be placed upon undertaker's boards or in wooden re-

ceptacles which have been cleaned with disinfecting solutions, lest they absorb minute amounts of arsenic, corrosive sublimate, or other poison, these substances being very common constituents of preserving fluids and disinfectants; the organs should be placed in glass or porcelain-lined dishes only, and the physician should see that these dishes were previously properly cleaned. The autopsy should always be performed in the presence of one or more medical witnesses.

Laying to one side all cases of death from suspected violence in which there is no suspicion of the death as having been caused by a poisonous substance, we recommend the following details of a method for conducting an autopsy which are taken from the work of Dr. Luff, to which we have previously referred:[31]

"If there is any clue as to the cause of death, the cavity supposed to be implicated should be opened first, otherwise the order given below should be followed. In all cases, however, it is important that all the cavities of the body should be opened and their contents examined; since, although a cause of death may be found in the first cavity, yet a counsel defending a prisoner could argue that an earlier cause of death might have been found in one of those left unopened.

"To open the body the operator should stand on the right side of the cadaver, and should make an incision in the middle line, through the skin and fat, extending from the chin to the symphysis pubis. The abdominal cavity should be opened first, and notes should be made of the relations and appearances of the viscera as seen *in situ,* and of any abnormal conditions of the peritoneum. The position of the diaphragm should then be ascertained on either side by passing the index finger of the right hand to the roof of the diaphragm in the midclavicular line, and then pressing the finger towards the front of the thorax so as to ascertain the height by reference to the ribs.

"After opening the thorax, the pericardium should be laid open along its lower and right borders, and notes should be made of the degree of distention of the right auricle and the ventricles, and the state of the serous membrane. Abnormal collections of serum, blood, or other liquid found in any of the cavities of the body should be carefully collected by a syringe or otherwise, and measured in a graduated glass vessel.

"Before proceeding further with the examination of the thorax and abdomen, it is desirable to expose the surface of the brain, so that the degree of congestion of its vessels may be ascertained before the great

[31] Op. cit. Vol. I. pp. 88 *et seq.*

vessels in the thorax or abdomen are cut. To effect this the skullcap is exposed by making an incision down to the bone from the root of the left mastoid process to the corresponding point on the right side, and then peeling from the skull the frontal and occipital flaps of the scalp. During this proceeding note should be made of any abnormal extravasations of blood that may be found, and careful search should be made for fractures. If a fracture be found, it should be followed to its whole extent, and any unusual thinness of bone should be noted. To remove the skullcap the outer table of the skull is sawed through on either side along a line passing anteriorly from a point an inch above the root of the zygoma to just above the frontal eminences, and posteriorly to a point about an inch above the external occipital protuberance. The separation of the skullcap is carefully completed with the chisel, precautions being taken to avoid fissuring the bones. The dura mater is then cut through on a level with the edge of the bone, except in the middle line behind, and is turned back so as to expose the surface of the pia mater. In infants under a year old the bones of the skull and the dura mater can be divided together by a strong pair of scissors. The degree of distention of the superficial cerebral veins is to be noted, and the examination of the abdominal and thoracic cavities should then be proceeded with.

18. Examination of abdominal cavity.—"The surface of the intestines should first be carefully inspected and palpated, and the condition of the mesenteric glands ascertained. The stomach should then be removed by placing a ligature round the lower end of the œsophagus, and a double one at the beginning of the duodenum, and dividing the œsophagus above its ligature, and the duodenum between the two. The stomach should then be placed upon a clean porcelain dish, and opened along the lesser curvature, care being taken that none of the contents are lost. The contents are poured into a clean jar, and the inner coat of the stomach is examined. Note should be made of its color, and the surface should be examined by the aid of a lens for particles of leaves, berries, coloring matter, crystals, etc., any of which, if found, should be examined under the microscope. Any signs of inflammation, ulceration, corrosion, or perforation should be noted. The œsophagus should then be removed and opened, and any pathological conditions, or signs of corrosive or irritant poisoning, should be noted. The small and large intestines are then to be separately ligatured, removed, and examined in a similar way to the stomach; note should be made of any pathological conditions, such as ulcers, perforations, growths, and areas of inflammation. The presence or

absence of feces in the lower bowel should be recorded. The liver, spleen, pancreas, kidneys, and suprarenal bodies should be removed and weighed and examined.

"The examination of the pelvic organs requires special care in cases of suspected criminal abortion. The peritoneal surface of the uterus, and the upper portion of the posterior surface of the vagina, are carefully examined for perforating wounds, and the vulva is also carefully inspected. The ligaments of the pubic symphysis are then divided with the cartilage knife, and the lower limbs are forcibly separated in order to cause the pubic bones to come apart, and so give room for the separation of the pelvic organs, which are then removed along with the skin of the perineum and vulva. The bladder is to be opened and examined. The vagina is then cut through with blunt-pointed scissors along the middle of its anterior surface, and its interior and the os uteri carefully inspected. The cervix and the body of the uterus are then cut open by a continuation of the incision into the vagina, and their interiors carefully examined. The ovaries should be opened by clean cuts, and notes made of any corpora lutea that may be present.

19. Examination of thoracic cavity.—"The cavities of the heart should be examined before it is removed, and the size of the tricuspid and mitral orifices should be tested by the fingers. After the removal of the heart, the mitral and aortic valves and the endocardium are thoroughly exposed and any abnormalities noted. The lungs and their pleural surfaces should be examined previous to removal, and evidence of aeration, collapse, or atelectasis carefully looked for. The lungs, trachea, larynx, floor of the mouth, and tongue should be removed together, and submitted to careful inspection.

20. Examination of cranial cavity.—"The brain is removed in the usual way, the spinal cord being cut as low down as possible, in order to facilitate the subsequent removal of the remainder of the cord. The brain should then be placed on its ventral aspect, and transverse and vertical sections made through its substance. The sections should not pass quite through the brain, so that the subdivisions may be easily replaced and held together, in order to facilitate the accurate location of any lesion that may be found.

"The spinal cord may be removed either from the front by the use of Schmidt's chisels, or from behind. After the surface of the cord has been examined, a series of transverse sections should be made at short intervals from end to end, in order to examine its substance.

21. Joints and bones.—"The first metatarsal-phalangeal, and other

joints should be opened to ascertain whether any deposits of sodium urate are present in the cartilages. If necessary, the bones should be examined by freeing them from the soft parts, and then making longitudinal sections with a sharp saw. Evidences of rickets and syphilis should be looked for. When the examination is made several days or longer after death, changes due to decomposition must be allowed for. If the corpse has been some months in the ground or in water, the formation of adipocere will mask the normal appearances, and may be mistaken for tubercular caseation.

22. Instruments to be used.—"The operator should be provided with a pair of scales, a graduated glass measure, some clean glass jars, an ordinary dissecting case, two pairs of scissors,—one pair sharpened and the other blunt-pointed,—two full-bellied section knives, a cartilage knife, a saw with a movable back, chisel and mallet, bone-forceps, a pair of bowel scissors, a blunt-pointed bistoury, a straight narrow-bladed knife for removing the tongue and floor of the mouth, and a knife with a broad blade about eighteen inches in length for making sections of the brain and other solid viscera. A packing needle and twine are needed for sewing up the body.

23. Preservation of viscera for analysis.—"It is the duty of the medical man to secure and seal vomited matters carefully, as well as the liquids and viscera taken from the body of the deceased, in clean vessels, and to affix a label to each vessel stating the nature of the contents, the name of the deceased, and the date of removal; the signature of the medical man should be added to every label. This is necessary, as the law insists that proof of the identity of the various articles should be forthcoming. The vessels in which the viscera are secured should be wide-mouthed glass jars, which should be thoroughly cleaned out by being first scoured with sand, then rinsed with strong hydrochloric acid, and afterwards washed with water. No antiseptic should be added. The vessels should be covered over with oil-silk, which should be tied down and a seal affixed to the string. Calico, linen, and paper of all sorts should not be employed for covering the vessels, since these articles may have arsenic or lead in the dressings that have been used in their preparation.

"The jars should be sealed with the private seal of the medical man who has made the necropsy. These jars should be delivered personally by the medical man to a responsible party, to whom a list of the jars and contents should be given, a similar list being retained by the medical examiner. If the jars and their contents have to be kept,

they must be locked up in a cool place. No preservative should be added to the contents of the jars.

"If a chemical analysis for poison is to be made of any of the organs, they should be placed by the physician himself in perfectly clean glass jars; glass preserve jars with a glass or porcelain-lined cover are suitable for the purpose, and can always be obtained in the country or city. Each organ should be placed in a jar by itself; for instance, the stomach in one jar, its contents in another, the intestines in another, contents of intestines in a fourth, the liver in a fifth, the kidneys in a sixth, the brain in a seventh, etc., etc. The organs which should be saved for chemical analysis are, in the order of their importance as follows: Stomach, contents of stomach, liver, intestines, contents of intestines, kidneys, brain, heart, spleen, and urine if there be any; in some cases it is important to save portions of the muscular tissue, and in others a part of the lungs. In some cases it is wise for the physician making the examination, to divide each of these substances into approximately two equal parts, the parts to be kept in separate jars, one to be given to one chemist for preliminary analysis, and the other to be retained by the physician himself, in case it may be necessary to have the analysis confirmed by another chemist, as is usually the case in trials for murder by poison. These jars containing the organs should be closed and sealed by the physician himself, the seal to be stamped with a private stamp. They should then be locked up until they are to be delivered to the chemist. It is better that the organs be placed under double lock, one key to be taken by one person and the other by another, so that neither alone has access to the organs; this is, of course, not necessary, if one person possesses the key, and another has possession of the stamp with which the seals have been stamped. When the jars are to be sent to a chemist, they should be sent by messenger, preferably two messengers; since, in the event of the investigation resulting in a trial for murder, the identity of the organs cannot be lost by the death of the messenger. The organs should never be sent by express, since it is, in that case, impossible to preserve with absolute certainty the identity of the organs.

[For precautions in regard to exhumations, reference is made to page 575, *infra,* and to details stated in the Thayer Case, Appendix, p. 791.]

24. Exhumation.—"In the case of the exhumation of a body, it should be properly identified by the friends or relatives of the deceased, before removal from the coffin, at which time, also, the medical inspectors should view it. Exhumations are most frequently made

in cases of suspected poisoning. In such cases, the stomach, intestines, liver, spleen, and kidneys should be removed and placed in clean glass jars. If a metallic poison be suspected, the shaft of the femur should be removed. If the body has been buried a long time, or if the coffin be decayed, a small portion of the surrounding earth should be taken away for chemical examination."

These details are particularly referred to because, in England and in other foreign countries, any careless omission is especially noted in trials for murder; and the ends of justice are protected by strict adherence to their requirements. The editors of the present edition of this work would especially call the attention of our readers to the report and analysis of a noted trial within the last two years in our own country. See in Appendix to this volume, Manson Case. In this case as reported, the medical evidence, in both trials before the court, was presented to show that the medical men who conducted the necropsy of the victim who died from poisoning by arsenic, as the verdict was given, sterilized the glass jars containing the viscera of the poisoned woman, but their method of sterilizing was not particularly investigated. Inquiry should be made as to whether the substances used for sterilizing may not have some ingredient which has a poisonous character.

25. Investigation of causes of death by post-mortem examination.—It will be well to consider here in brief the question of the investigation of the cause of death by post-mortem examination. The theory that professional opinion of a competent and well-educated medical man shall prejudge the case without dependence upon a jury of unprofessional men is fast gaining ground in the department of forensic medicine, and, as already stated, has given origin to a new form of process in the states of Massachusetts and Connecticut. The question, then, of pathology becomes an important matter to be clearly understood in all judicial inquiries, and should demand more consideration in books on toxicology than has heretofore been given to it. Without, however, attempting to present a treatise on pathology, which would be entirely incomplete and out of place in this work, there are certain facts which should be clearly recited.

First. How should a post-mortem examination be conducted? To answer this question the following suggestions are incorporated from the Transactions of the Massachusetts Medico-Legal Society.[32]

[32] Vol. I, p. 139, Riverside Press, Cambridge, 1879.

"SUGGESTIONS FOR THE MEDICO-LEGAL EXAMINATION OF DEAD BODIES.

REPRINTED FROM THE REGULATIONS ADOPTED BY THE CROWN OFFICE AT EDINBURGH, SCOTLAND, TO BE OBSERVED IN CRIMINAL AND OTHER INVESTIGATIONS.[33]

"In the following suggestions we have not attempted to lay down full and minute instructions for the complete examination of dead bodies in all possible medico-legal cases; because such a plan would involve a statement of many long details, of which every medical man ought to acquire a competent knowledge in the course of ordinary study and practice, and especially in performing ordinary dissections for discovering the signs of disease. We have thought it of more importance to confine our attention to points which are of essential consequence in judicial investigations, or which are apt to be neglected in common dissections.

"It will be remarked that we propose to turn the attention of the medical inspector to some points which are often inquired into,—not by him, but by magistrates or other official persons not of the medical profession,—such as the place where the body is found, its position when first seen, surrounding objects, the clothes, the history of cases of suspected poisoning, and the existence of poison in a house, etc. This we have been led to do, because we have had occasion to observe that on such points important articles of evidence have been overlooked, owing to the absence of a medical man, to whom alone their importance would have been apparent. On the whole, however, such matters would probably be best investigated by a law officer with the aid of a medical man.

I. *General Duties.*

. . . . . .

"(4) It is desirable that the medical inspectors shall have an op-

[33] Through the courtesy of Prof. Douglas Maclagan, of Edinburgh, the Massachusetts Medico-Legal Society has received a copy of the code of regulations governing the proceedings of legal and medico-legal officers in Scotland, in their criminal and other investigations. This code includes an appendix, containing, among other matter, a series of suggestions for the medico-legal examination of dead bodies; these "suggestions," prepared by three Scotch professors and medical jurists of distinguished and world-wide reputation, are so comprehensive in their character, and are so well adapted to aid Massachusetts medical examiners in the performance of their official duties, that the committee on publications takes pleasure in fulfilling the society's instructions to reprint them in this form. Certain paragraphs not belonging to toxicology are omitted.

portunity of viewing the body before it is undressed, or moved from the spot where it was first found. If the body has been previously removed or meddled with, they ought to inform themselves accurately as to its original position. In many cases, it is material that they personally visit the place where it was first seen; and they should inquire minutely into all the particulars connected with the removal of it.

"(5) In cases where the body has been buried, and disinterment becomes necessary, it ought not to be removed from the coffin except in presence of the inspectors. In any case where subsequent investigations may involve a search for poison in an exhumed body, one or two pounds of the soil immediately above the coffin should be secured for analysis.

"(6) When a considerable period has elapsed between death and disinterment, the inspection must, in all cases, be proceeded with, although the body be found in a state of decay, unless the inspectors can positively say that the progress of decay is such as to render the examination nugatory in relation to its special objects. The degree of decay which will justify such an opinion will differ with a variety of circumstances which cannot be properly specified here. It may be observed, however, that, where injuries of the bones are to be looked for, or the traces of certain poisons, it is scarcely possible to assign the limit at which an inspection must of necessity be fruitless. It is of moment to remember that the internal organs are often in a great measure entire, although the external parts are much decayed. The dissection, where the body is putrid, will be rendered less annoying to those present by sprinkling the surface of the body with a solution of chlorid of lime (one part in forty), or of carbolic acid (one part in thirty); but none of these disinfectants should be applied after the dissection is begun, especially in cases where there may afterwards be an examination for poisons.

"(7) No one should be allowed to be present at the examination out of mere curiosity; and we recommend that everyone not engaged in the inspection, but who is in attendance to give information, or for any other purpose, and who may afterwards become a witness, should remain in an adjoining room. The medical inspector often furnishes good tests of the value of other evidence in the case; and it is therefore desirable that the general witnesses should not have an opportunity of knowing what is observed in the dissection of the body.

"(8) The examination and dissection of the body should not, if

possible, be commenced without sufficient daylight in prospect to allow the whole inspection to be made without artificial light.

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"(13) When any portions of the body, or any substances found in or near it, are to be preserved for further examination, they ought never to be out of the custody of the inspectors or of a special law officer. They must be locked up in the absence of the person who keeps them. When they are to be transmitted to a distance (which is to be done by the hands of an authorized individual, not by sending them by a public conveyance), the vessels containing them should be secured and sealed upon the spot. A label ought to be attached to each vessel, bearing a list of the articles therein contained, and dated and signed by the inspectors. Narrow-necked vessels and bottles may be secured by corks and wax impressed by a seal; wide-mouthed jars and other vessels, if large corks cannot be had, are best secured by tying them over with sheet gutta-percha, fortified, if requisite, with leather. It is important to observe that the covers are securely tied and sealed, so that the coverings cannot be removed and replaced without either cutting the strings or breaking the seals.

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III. *External Aspect and Examination of the Body.*

"(16) The importance of the external examination, and the particulars to be chiefly attended to in performing it, will vary in different cases with the probable cause of death. It comprehends an examination: 1. Of the position of the body when found, as well as of all external injuries or marks presented by it. 2. Of the vicinity of the body, with a view to discover the objects on which it rested, or from or upon which it may have fallen; marks of a struggle; signs of the presence of a second party about the time of death or after it; weapons or other objects, the property or not the property of the deceased; the remains of poisons; marks of vomiting; and where marks of blood are of importance, and doubts may arise as to their really being blood, the articles presenting them must be preserved for further examination. 3. Of the dress, its nature and condition, stains on it of mud, sand, or the like, of blood, of vomiting, of acids or other corrosive substances; marks of injuries, such as rents or incisions. Where injuries have been inflicted on the body, care should be taken to compare the relative position of those on the body and those on the clothes; and where stains, apparently from poison, are seen, the stained parts are to be preserved for analysis. 4. Of ligatures, their material and kind, as throwing light on the trade of the person who

applied them, the possibility or impossibility of the deceased having applied them himself, their sufficiency for accomplishing their apparent purpose, etc.

"(17) Inspectors will commence the examination of the body itself by surveying the external surface and openings. Before cleaning it, they will examine it on all sides, not neglecting the back, as is often done, and look for marks of mud, blood, ligatures, injuries, stains from acids and the like, foreign bodies, or injuries within the natural openings of the body, namely, the mouth, nostrils, ears, anus, vagina, and urethra. If there are impressions of finger marks, they will consider which hand produced them. If there be any doubt about stains being blood, the skin presenting them must be preserved for analysis. If there be acid stains, or other probable remains of poison, or any foreign matter, the nature of which may require to be determined by analysis, these must also be preserved. The ordinary places for the impressions of ligatures are the neck, the wrists, the ankles, and the waist. The degree of warmth of the trunk and extremities, the presence or absence of cadaveric rigidity, and whether it exists equally in the upper or lower extremities, should be noted in this stage of the proceedings; in other cases, the progress of putrefaction, as indicated by the odor of the body, the looseness of the cuticle, the color of the skin and the formation of dark vesicles on it, the evolution of air in the cellular tissue, the alteration of the features, the softness of the muscles, the shriveling of the eyes, the looseness of the hair and nails.

"(18) In this part of the examination, it will sometimes be necessary to observe the particulars by which the body may be identified. These are numerous; but the most important are the stature, the degree of plumpness, the size and form of the nose and mouth, the color of the eyes and hair, the state of the teeth, warts, nævi, deformities, scars of old abscesses, ulcers, and wounds.

"(19) The body is next to be washed, if necessary, and the hair of the head shaved, or at least closely cut; and a thorough examination of the whole integuments is to be made. At this stage, the inspectors will look particularly for the appearance of lividity, noting its chief seat and its relation to the posture in which the body was found; for impressions on the skin of objects on which it had rested; for marks of injuries, more especially contusions, taking care to ascertain their real nature by making incisions through the skin; for marks of disease, such as eruptions, ulcers, and the like; for marks of burning; for marks of concealed punctures in the nostrils, mouth, external opening of the ears, the eyes, the nape of the neck, the armpits, the

anus, the vagina, and beneath the mammæ or scrotum; in infants, also in the fontanelles and the whole course of the spine. At this stage, wounds and other injuries should be carefully examined according to the directions given in Division V. *infra*. Where the injury may have caused loss of blood, the presence or absence of pallor in the skin, lining membrane of the mouth and the gums, ought to be noted.

IV. *Dissection or Internal Examination of the Body.*

"(20) In commencing the dissection of the body, it must be laid down as an invariable rule that all the great cavities should be examined, and also every important organ in each, however distinctly the cause of death may seem to be indicated in one of them. It is right to examine the cavity of the spine, and, at all events, its upper portion, in any case where an unequivocal cause of death has not been discovered elsewhere.

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"(22) The inspectors should begin with that cavity over which there is a wound, or other mark of injury. Or, if there be an injury on the extremities, the dissection ought to commence there. In the absence of any such guide, that cavity should be taken first where the circumstances of death, so far as they are ascertained, may lead the inspectors to expect unusual appearances. In other cases it is best to lay open the chest and abdomen, to take a general survey of the parts exposed without disturbing them materially; or to proceed to the head, which may be examined thoroughly in the first instance; afterwards to examine carefully the chest and belly; and the spine may be reserved till the conclusion. Wherever unusual appearances are discovered in the first cursory survey, the anatomical examination ought, in general, to be begun there.

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"(29) It is necessary to examine the pharynx and gullet, the larynx, trachea, and its greater ramification, the lungs, the heart, and the great vessels with particular care, because here are most frequently found the causes of sudden natural death. In examining the heart, each auricle and each ventricle ought to be laid open by an independent incision of its parietes, and this should not intersect any of the valvular openings of the septum cordis.

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"(31) In inspecting the organs in the chest, a cursory examination should be first made by turning them over, ascertaining the na-

ture, and measuring the quantity of effused fluids, feeling for tumors or other diseases, and opening the pericardium to obtain a view of the heart. The most convenient course to pursue next is, without moving the heart from its place to lay open its several cavities, in order to judge of the quantity and state of the blood in both sides of that organ, and then to remove the whole organs in the chest, namely, the lungs, heart, and gullet, together with the parts dissected downward from the throat, in one mass, and to examine them in detail on a table. But previously a ligature should be applied on the gullet, just above the cardiac orifice of the stomach.

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VI. *Examination in Cases of Poisoning.*

"(42) In examining a body in a case of suspected poisoning, the inspectors should begin with the alimentary canal first, tying a ligature round the gullet above the cardiac orifice of the stomach, and two round its pyloric end; then, removing the stomach and whole intestines; next, dissecting out the parts in the mouth, throat, neck, and chest in one mass; and, finally, dissecting the gullet, with the parts about the throat, from the other organs of the chest. The several portions of the alimentary canal may then be examined in succession.

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"(44) The medical inspectors may afford most important aid to the law officers in investigating the history of cases of supposed poisoning. For this purpose minute inquiry should be made into the symptoms during life,—their nature, their precise date, especially in relation to meals or the taking of any suspicious article, their progressive development, and the treatment pursued. It is impossible to be too cautious in collecting such information; and, in particular, great care must be taken to fix the precise date of the first invasion of the symptoms, and the hours of the previous meals. The same care is required in tracing the early history of the case, when the inspector happens to visit the individual before death; and, if suspicions should not arise till his attendance has been going on for some time, he ought, subsequently to such suspicions, to review and correct the information gathered at first, especially as to dates. All facts thus obtained should be committed to writing.

"(45) Besides inspecting the bodies and ascertaining the history of the case, the inspectors may afford valuable assistance to the law officers in searching for suspicious articles in the house of the deceased. These are suspected articles of food, drink, or medicine; the

vessels in which they have been prepared, or afterwards contained; the family stores of the articles with which suspected food, etc., appear to have been made. All such articles must be secured according to the rules in section 13 for preserving their identity. In this examination the body, clothes, bedclothes, floor, and hearth should not be neglected, as they may present traces of vomited matter, acids spurted out or spilled, and the like.

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"(48) The whole organs of the abdomen must be surveyed, but particularly the stomach and whole tract of the intestines, the liver, spleen, kidneys, bladder, and, in the female, the uterus and its appendages. The intestines should in general be slit up throughout their whole length; and it should be remembered that the most frequent seat of natural disease of their mucous membrane is in the neighborhood of the ileocæcal valve, and that, next to the stomach, the parts most generally presenting appearances in cases of poisoning are the duodenum, upper part of the jejunum, lower part of the ileum, and rectum.

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"(51) The stomach and intestines should be taken out entire, and their contents emptied into separate bottles. If the stomach or part of the intestines presents any remarkable appearance, examination may be reserved, if convenient, till a future opportunity; but in every instance, it must be preserved and carried away, as it may itself be an important article for analysis. The throat and gullet may be examined at once, and preserved with their contents, which, if abundant, may be kept apart in a bottle. In addition to the alimentary canal and its various contents, portions of the solid organs of the body ought to be secured for analysis. The most important are the liver, spleen, and kidneys. A part of the liver, at least a fourth part, should be secured in every case of supposed poisoning; and in cases where the fatal illness has been of long duration, and therefore only traces of the poison may remain in the body, the whole liver, the spleen, and both kidneys should be secured. A portion of the blood, especially when the odor of any volatile poison is perceived, should be at once put into a bottle, closed by a good cork or stopper.

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R. Christison.
James Syme.
Douglas Maclagan."

26. Rigor mortis or cadaveric rigidity.— Rigor mortis or cadaveric rigidity is due to a post-mortem irritability inherent in the muscles,

the effect of which is to produce a firm and continuous contraction in their length; usually the contraction first commences in the muscles of the jaw and thence extends downwards. In the more robust individuals this cadaveric rigidity sets in earlier than in those who have died from a long and lingering illness.

It is not reasonable to attempt to lay down any specific rules which govern either the early appearance of this post-mortem sign, its duration, or its time of disappearance. It is still more unreasonable to attach any very great specific value to the time of its appearance, its duration, or time of disappearance after death as a means of determining the kind of poison used for destroying life. In spite, however, of this view of the question, the conditions of cadaveric rigidity have a certain circumstantial value in connection with symptoms which precede death by poison. In general terms this statement should be allowed, *viz.*, that the appearance of post-mortem rigidity depends upon the inherent irritability of the muscular tissue. When death has been preceded by violent exercise of this inherent muscular irritability, the post-mortem rigidity sets in early and lasts but a short time, because the muscular irritability has been more or less exhausted. By way of illustration the following observation was made by the writer:[34] A brakeman who had been in muscular activity just before death, and was coupling together two railroad coaches having platforms of unequal height, was suddenly compressed by the backward motion of the engine. Judging by the circumstances, the platform of the forward car struck him in front just below the diaphragm, whilst the platform of the other car struck his back about four inches higher. Death was instantaneous. "I arrived about twenty minutes after the accident, and found the body of the man (which had been disengaged from the position of the accident at the time of the compression) lying upon its back in a state of commencing post-mortem rigidity." The muscular rigidity was only present in the head and trunk, not yet having reached the lower portions of the body, though six hours afterwards the whole muscular system was in a state of strong contraction. When first seen after the accident, the right arm was extended upwards in a state of semiflexion, and the left arm was stretched out from the body, slightly flexed at the elbow joint, in fact, in just the position assumed for dropping the connecting pin into its socket with one hand, while the other was held up to signal the conductor when the car was connected. This single observation illustrates the question of an early

[34] Reported in Boston Med. and Surg. Jour. Dec. 4, 1873.

appearance of post-mortem rigidity, which is sufficiently common in military surgery,[35] especially after wounds apparently instantaneously fatal, as of the head and heart. Dr. Brinton remarks: "I have seen bodies rigid in their last attitude, twenty-four, forty-eight, and in one instance sixty, hours after death. M. Armand, at Magenta, saw this rigor twenty-four hours, and M. Perier, at Alma, forty-eight hours, after death. The fact of the prolonged continuance of this rigor would seem to militate against the idea of its tetanic nature, and of its being followed in turn by flexibility and by rigor mortis proper." On the other hand, probably from observations made upon persons and animals killed only by poisons, M. Tardieu[36] is misled into making the supposition that convulsifying poisons induce a specific effect upon the time of appearance and permanence or duration of post-mortem rigidity, while those which relax the muscular system before death have a contrary effect.

In reference to this question we again quote from Dr. Vivian Poore: "Rigor mortis is due to the coagulation of muscle plasma. As a rule the muscles remain in the position in which they were at death. But that is not quite true. . . . The time of onset of the rigor mortis varies, but as a rule it is said to begin five to six hours post mortem. But this is so variable that there can be no rule at all. Beginning, as a rule, in the jaw, it spreads to the face, neck, trunk, and limbs. It would appear, practically, that the time of the onset of rigor mortis bears a direct ratio to the irritability of the muscle at the time of death. If the muscle is in good condition, with its normal irritability, rigor mortis sets in slowly; if the muscle is exhausted, rigor mortis sets in early. Now, in persons who die while undergoing great muscular exertion, the muscles are fixed sometimes at the moment of death apparently. Whether this be precisely analogous to rigor mortis, or whether it be a spasm occurring at death which continues until that spasm is fixed in rigor mortis, is a debated point. As a practical fact, when the muscles are exhausted by excessive work at the moment of death, rigor mortis or cadaveric spasm comes on instantly. The hare which has been coursed to death stiffens very quickly because the muscles are exhausted, and rigor mortis sets in immediately and passes off quickly. The coursed hare may therefore be eaten by the epicure earlier than a shot hare, because it is fit to eat earlier. So, also, on the field of battle after forced marches, or when

[35] See an interesting communication on "Instantaneous Rigor as the Occasional Accompaniment of Sudden and Violent Death," by John H. Brinton, Am. Jour. of Med. Sci. Jan. 1870.

[36] Op. cit.

a man is killed while fighting for dear life, and putting forth a quite unusual amount of muscular energy, it is found that he remains fixed in the position in which he was when he was killed. That is apparently due to the exertion, and possibly also to the position in which he was when killed. A man shot through the head is very often fixed in the position in which he fell, and I take it the evidence is perfectly good that soldiers have been found grasping the rifle and in the position adopted for firing, perhaps in readiness to shoot over an earthwork. There is a case which comes from Germany of a man who got out of a train at a railway junction, and went across the tracks to the refreshment room; he came out of the refreshment room with a sausage in one hand and a sandwich in the other, and on going back to the carriage his head came between the buffers of opposing carriages, and he was killed instantly. When an examination was made, the sausage and the sandwich were tightly grasped in the hands. That was due to some spasm of the muscles caused by the shock to the brain. So it happens sometimes in death from lightning that rigor mortis comes on suddenly. The sooner rigor mortis comes on the sooner it passes off; that is the rule. John Hunter came to the conclusion that in death from lightning rigor mortis did not set in. The reason for that belief was probably that it set in early and passed off early, and at the time of inspection it had ceased. And so, when patients die of exhausting diseases, with presumably little muscular irritability left, the rigor mortis sets in early and passes off early. That is one of the difficulties with people who die gradually. In those cases rigor mortis either does not come on, or it is badly developed and passes off quickly, and the people think that the body is not dead because it has not got stiff. . . . I must remind you that after excessive exercises,—we all must have experienced it after a walk of unusual length,—when, after a rest, we rise to continue our journey, our walking is labor and sorrow, because the muscles are all stiff. . . . You know that when we are very intent upon something, we put forward an enormous amount of muscular power, very much more than is necessary. This probably occurs in battle. When a muscle is severed from the nerve centers, rigor mortis is slow in coming on."

For an explanation of the cause of rigor mortis, or cadaveric rigidity, we quote from Dr. Luff:[36a] "Experiment shows that the seat of rigor mortis is in the muscles, for the rigidity disappears on their division. During the stage of rigor mortis the muscles

[36a] Op. cit. Vol. I. p. 27.

retain the precise position they occupied at the time that rigidity supervened. The cause of the rigidity is the coagulation of the myosin, or muscle fibrin within the sarcolemma of the muscular fibers. With regard to the actual causation of the coagulation of the myosin, one view that has long held is that it is effected by means of weak acids. Sarcolactic acid is being constantly formed during life, and as constantly removed from the muscles and neutralized by the blood circulating through them; but after death, during the period of life that remains to the muscles, and while they are still flaccid and contractile, acid products continue to form and accumulate in the muscles, so that the muscles in a condition of rigor mortis have a strong acid reaction, due to the presence of sarcolactic acid, and possibly also of glycerophosphoric acid. The rigidity of the muscles exists in two stages,—an early stage and a later one; time simply determining the lapse of the former into the latter. In the early stage of rigidity the myosin is only partially coagulated; and if, while the muscle is in this stage, fresh arterial blood is made to flow through its vessels, the semi-coagulated myosin is dissolved, and it is then found that the muscles are still irritable to electrical stimuli (Brown-Sequard). The latter stage is due to the complete coagulation of the myosin; and when this stage is reached, there is a permanent loss of irritability of muscles to electrical or mechanical stimuli."

The literature of the action of poisons on rigor mortis is voluminous and contradictory,[37] and opinions based upon conclusions vouchsafed by French authorities should be taken with great caution, and only after careful study of the facts related by them in support of these conclusions.

M. Rondeau[38] draws from his own experiments, which seem well founded and carefully performed, the following conclusions:—

"1st. Contrary to the opinion of Hunter, muscular rigidity occurs after death by lightning; its appearance and duration are very variable.

"2d. Chloroform, whether by its introduction into the stomach or lungs, appears to prolong the duration of rigor mortis, though it has no apparent influence upon its production.

"3d. Phosphorus has no definite action upon the conditions of rigor mortis.[39]

[37] See a thesis, No. 396, 1880. Paris, by Pierre Rondeau, Etude Expérimentale sur la Rigidité Cadavérique au Point de Vue Medico-Legale.

[38] Op. cit.

[39] Tardieu presents examples which seemed to him to suggest a law of cause and effect, but does not insist upon its terms.

"4th. Morphin and laudanum—no facts worthy of note.

"5th. Cyanid of potassium appears to have a definite effect upon the time of appearance and duration of post-mortem rigidity." It usually appears in a few hours, and persists for two or three days.[40]

"6th. Arsenic does not modify post-mortem rigidity.

"7th. Oxalic acid apparently hastens the appearance of rigidity, which is preserved for a relatively long time."

8th. . . . Does not relate to the question of rigidity.

"9th. Salicylate of sodium apparently causes the early appearance and a very rapid disappearance of rigor mortis.

"10th. Sulphate of quinine, according to Niderkorn, causes an early and somewhat prolonged appearance of rigor mortis, during which decomposition rapidly goes on."

11th and 12th have no conclusive bearing upon this subject.

"13th. Death by drowning does not modify rigidity.

"14th. A fetus born dead is in a state of muscular rigidity."[41]

We would refer our readers to the detailed descriptions on the action of each poison for any known influence it may possess relative to the hastening or retarding effects upon the period of post-mortem or cadaveric rigidity.

With regard to the natural disappearance of rigor mortis, we would only venture the remark that we should not expect this to occur until after the setting in of the ordinary post-mortem putrefactive changes in the animal tissues.

27. Sources of error arising from natural changes in the body after death, in general.— Having thus shown the chief means of distinction between the effects of poison and of some of the natural diseases to which the human frame is subject, it only remains for us to point out some sources of error to which the natural changes taking place in the body after death may give rise. This important subject is one which has received but little attention at the hands of medical jurists, but there can be no doubt that the natural appearances of those parts of the body usually inspected after death, where poisoning has been attempted or is alleged, are often mistaken for pathological changes induced by the administration of poison. Since the publication of the first edition of this work, the knowledge by pathologists of the marks of disease, as distinct from those due to natural post-mortem appearances, has become so well established that the medical witness, whether

[40] See also a paper published by the writer in the Boston Med. and Surg. Jour. Aug. 23, 1866.

[41] The experiments related by the above author made under variable conditions of temperature from (6° C.) 42° F. to (23° C.) 64° F.

he be a trained expert or a well-qualified practitioner, ought to be able to show clearly whether the post-mortem appearances in a given case are natural or abnormal, whether due to natural causes or the marks of disease: the post-mortem appearances due to natural causes would not be likely to mislead in an investigation of cases of suspected poisoning.

Among later writers, Dr. Luff[41a] states that "the cooling of the body after death depends upon the following causes:—

"(1) The cessation of certain chemical processes in the body.

"(2) On radiation of heat; thus, a body will cool more rapidly in the open air than in a dwelling, more rapidly on the floor than in bed, more rapidly in a large apartment than in a small one.

"(3) On the conduction and convection of heat by the medium or substance in contact with the corpse; for instance, a body will cool more rapidly in water than in air, provided, of course, the water is cold; more rapidly if naked than if covered with clothing; more rapidly in the case of a lean or emaciated body than in the case of a fat or corpulent one."

The length of time elapsed after death of murdered persons may form an important link in establishing the criminality of the accused murderer. The case reported by Taylor, of the murder of Mrs. Gardner, is a case in point. The time after death was a point of importance. The husband of the woman was accused of having caused her death. Her body when found was lying on the floor, covered with a flannel petticoat and a chemise. The upper limbs were cold and rigid, and the trunk was cold. The only warmth was in the abdomen, which was due to the woman's pregnancy. The prisoner was absent from home four hours. Proof was given that the woman had been dead more than four hours, probably ten hours or more. Counsel for the prisoner contended that the body might have become cold and stiff within the four hours that the prisoner was away from home, in order to establish his innocence. The jury declared otherwise and Gardner was convicted.

28. **Condition of the skin.**—Usually the surface of the body cools slowly, corresponding with that of the surrounding air, in from fifteen to twenty hours after death. The exceptions to this rule depend upon circumstances; such, for instance, as when the body is covered by clothing, or left naked with the surface exposed; or when death results from certain diseases, such as cholera, yellow fever, rheumatic fever, smallpox, cerebro-spinal meningitis, strychnin poisoning, tet-

41a Op. cit. Vol. I. p. 23.

anus, some brain diseases, and injuries to the brain substance. In most of these instances this unnatural post-mortem rise of temperature will be due to chemical changes in the body tissues, which, starting just before the death on account of active chemical decompositions, cause the result of heat, and will continue after death has set in. This action may be increased by the active spasms of muscular contraction which are especially illustrated in strychnin poisoning; this muscular contraction is associated with the evolution of certain organic acids being produced as the result of all muscular contractility; one of these acids we have before mentioned under the name of sarcolactic acid, which is the result of chemical action as a consequence of this muscular contraction.

According to the best observers, the rate of cooling of the body after death is greater during the first period under similar conditions—4° to 5° or more per hour—than during the last period, when it may be only a fraction of a degree per hour. If the question of determining the time elapsed after death is important in any medico-legal inquiry, the observer should invariably test the bodily temperature by means of thermometers placed in the armpit and inside the rectum. It must be remembered that the rate of cooling of the dead body will be influenced very much by the difference between the temperature of the body and that of the surrounding medium. It is a well-known fact that, after death from some of the above-named diseases, the bodily temperature may even rise, as, for instance, in cholera to 113° F.

There is a case recorded, in the year 1889, in the London Lancet, of a patient who died from an attack of uremia and a one-sided paralysis, and who had, just previous to death, received hypodermic injections of pilocarpin, followed by sweating. Nothing unusual was discovered for a few hours after death, during which time the body was washed and covered with sheets. Post-mortem discolorations were observed sixteen hours after death, and the sheets and bedclothes were saturated with sweat, which continued for eight hours; previous to this sixteen hours, the surface of the body was as usual after death.

29. Color of the skin.— In order to obtain a definite knowledge of the complexion of the skin, the surface should be clean. The effects due to atmospheric exposure during life will, of course, be apparent in those parts not ordinarily protected by clothing. A peculiar bronze hue, even if it extends to the mucous surfaces, does not always indicate disease of the suprarenal capsules, commonly known under the name of "Addison's disease," and, therefore, no conclusion should be drawn from the color of the skin only; it may be yellow-colored from

the presence of the bile in the circulation during life, and "if the jaundice be of long duration and very intense, the discoloration may be dark-yellow or even nearly black."[42] The use of nitrate of silver as a medicine during life produces a grayish or slate-colored complexion. A waxy-yellow color of the skin indicates a state of anemia during life, or may be the result of hemorrhage before death, or of blood poisoning from an abscess or other definite disease.

The changes of color resulting from post-mortem decomposition are (*a*) a greenish hue which appears earliest on that portion of the surface adjacent to the viscera; (*b*) a livid-reddish hue of the underlying portions of the body, or this discoloration may be seen in the subjacent tissue in the form of suggillations ("maculæ emortuales") or spots in which the blood, escaping from its vessels, lies in a noncoagulated form just beneath the external surface, or in a "boggy" tissue. Some of these spots may be due to the position of the body causing a natural gravitation of the blood, while others may be in consequence of the escape of the blood-coloring matter into the neighboring tissues. A discussion as to whether these spots are ecchymoses, or ante-mortem, will be more fully presented in the volume on surgical injuries. It is sufficient here to note that a post-mortem discoloration due to gravitation usually disappears on pressure, whilst that due to ante-mortem diffusion will not so disappear.[43] These post-mortem discolorations never give rise to elevation of surface as do those inflammatory spots which occurred during life.

Bruises made on the skin surface before death are generally easily recognized by the medical man; these are usually marked by an extravasation of blood into the surrounding subcutaneous tissue, which has a black and blue color. This discoloration, for a few hours after a blow, has a purplish and yellowish-green hue, gradually, in life, changing in the course of ten to twelve days after the injury into a purplish-black hue; in two or three days it becomes violet; after six days green; on the tenth day yellow; in about two weeks after the injury the skin generally assumes its natural color.

Dr. Luff states:[44] "Post-mortem stains are of a purplish or dull-red character; they at first impart a mottled appearance to the skin; later on, the individual patches coalesce and form large areas of discoloration. These patches generally terminate abruptly in the white skin, so that they possess a well-defined though irregular outline. . . . They are due to simple congestion of the capillaries, and

[42] Orth, op. cit.
[43] *Ibid.*
[44] Luff. Op. cit. Vol. I. p. 46.

not to sanguineous effusion. The position of these post-mortem stains may vary during the period that the blood remains fluid, if the body be disturbed. . . . The post-mortem stains, however, become permanent in position when coagulation of the blood has occurred, and after this has taken place no effect is produced upon them by altering the position of the body, nor are new stains formed; . . . although these stains appear on the most dependent parts, yet they do not occur on those parts of the body that are subjected to actual pressure."

30. Visceral decomposition.— The contents of the stomach may be acid from the fermentation of food, as, for instance, milk, which may soften the walls, and even digest them as has been before stated, and so mislead the observer to suppose that some corrosive poison had been administered previous to death; a chemical analysis would very readily solve the doubt, but if the digestion be due to a mineral acid, it would also corrode or char the stomach contents and adjacent tissues. Besides these changes due to the quantity and character of the contents of the stomach, which changes are mostly confined to the larger curvature of the organ, from the fact that the food lodges there, it must be observed "that the least degree of acidity produces a gray opacity; when greater, a sort of digestion of the mucous membrane itself follows, so that it is converted into a soft, slimy, transparent mass, which is easily scraped off, and then the submucous tissue, or muscular layer, is laid bare. Finally, the softening may go still further, and involve the muscular and serous coats, when the already described softening of the stomach results. If this occurs in a stomach free from blood, it is termed 'white softening;' if, on the other hand, the vessels are filled with blood, this is affected by the acid so as to present a brown or brownish-black color, the neighboring parts are infiltrated with the coloring matter, and a soft, more or less brown, mass results,—'brown softening' of the stomach. A dirty-green color is due here, as in most other organs, to actual putrefaction."[45]

In contrast with these appearances, the results of inflammatory processes in the stomach are quite distinct, and are carefully laid down in the latest works on pathological anatomy; these are acute and chronic catarrh, parenchymatous inflammation, and that of the submucous tissue. The parenchymatous form also occurs in poisoning from phosphorus and arsenic, and, as has been already observed, is associated with similar appearances of the tissues of other viscera. Therefore, in this form of general degeneration of organs observed

[45] Orth, op. cit.

at the autopsy in the case of a suspicious death, it would be advisable to make a chemical examination for poison.

Still we cannot omit commenting upon a certain post-mortem appearance which has been referred to in works on forensic medicine, especially in France, and ascribed to sudden and violent death by asphyxia, as well as by certain poisons. This appearance has been called subpleural ecchymosis. Dr. Legroux has recently investigated the nature of these hemorrhagic spots, and in a report to the Société de Médecine de France,[46] and also to the Congrès International de Médecine Legale of 1878, has presented this matter so much in detail that its omission here would not be admissible. Their special location seems to be on the free borders of the lobes of the lungs, especially the lower lobes, where the pleural surface glides over the diaphragm, on the surface adjacent to the diaphragm, or that which is contiguous to the lobes, less often on the convex anterior surface, and more rarely still at the apex of the lungs. The dimension of these ecchymotic spots is variable, from an almost imperceptible point to that of a lentil (*taches lenticulaires*), or of a ten-cent or twenty-cent silver piece. There may be only one or two, or as many as four or five, or even twenty, upon the whole pulmonary surface. These bloody spots are disposed separately on the different localities above mentioned without any particular relation to each other, or may be grouped in small numbers by islets of four or five, some of which may fuse into others; occasionally they are so small and numerous that the pleura has a marbled look, like the marbled wood which painters form by spattering paint from a brush on a neutral tint. If the lung be carefully examined, after its surface has been washed and wiped, it will be seen that neither pressure, washing, nor scraping can obliterate these spots; yet in animals freshly killed, if the lungs are artificially inflated, when the heart is cut into they are dissipated; if, however, the post-mortem examination be not made until some time after death, no artificial means can make these hemorrhagic spots disappear, and if the blood has lost its fluidity, and decomposition has progressed, they are even more distinct. They can be distinguished from the false ecchymotic spots so often observed in autopsies made some time after death, by the disappearance of these latter simply by inflating the lungs.

Recent writers on toxicology, among whom is Dr. Luff, have undertaken to explain the cause of these subpleural ecchymotic spots as a post-mortem change. We quote from the latter: "The production of

[46] Bull. tome V. 1879, p. 283.

post-mortem stains in the internal organs should be carefully remembered, as the simple effects of gravitation can produce a livid or red appearance of the dependent portions of the stomach and intestines. This staining or hypostasis has been especially noticed in the small intestine, particularly in those portions found in the cavity of the pelvis. This cadaveric congestion or hypostasis has long been known to exist in the lungs, where it affects, as a rule, the posterior fourth of each lung, and, consequently, there is a risk of attributing such a condition of the lungs to pathological causes. A similar cadaveric congestion of the stomach and intestines has not attracted such attention as the subject deserves, resembling, as it does to some extent, the effect produced by irritant poisons. Such a mistake may be avoided by noting the absence of inflammatory exudation, and the fact that, on stretching the opened intestines, the discoloration will be found absent in the divisions or folds lying between the discolored portions. If the body has been lying on its back, the posterior halves of the kidneys are usually gorged with blood; the veins of the pia mater at the posterior part, the lateral and occipital sinuses, and the veins in the pia of the cord, are often found filled with blood.

"The great importance of distinguishing these post-mortem stains from bruises inflicted during life is obvious. In the following table the distinguishing characters are given:"—

"DISTINCTION OF BRUISES FROM POST-MORTEM STAINS.

	Bruises produced during life.	*Post-mortem stains.*
Anatomical seat..	Effusion of blood from ruptured vessels into the subcutaneous tissues and true skin.	Congestion of the capillaries of the retemucosum above the papillæ.
Position.....	The seat of the injury.	The dependent parts of the body not actually subjected to pressure.
Appearance.....	Frequently have the shape of the instrument that inflicted the injury. The color is not generally uniform, and the bruise is usually elevated.	Irregular in shape, but with well-defined edges. The color is uniformly purplish, and the stain is not elevated.
Results of incision.	Effused blood, either in a clotted or fluid condition, is found.	No effusion of blood is found.
Changes by time..	Zones of color form around the edge, the centre always being the darkest. The colors are purple, violet, green, yellow, and lemon; they are dependent on the different degrees of oxidation of the effused blood.	No zones of color around the edge. The purplish color of the stain remains constant until putrefaction sets in."

When cut into, the true subpleural ecchymotic spot is found thin, and scarcely penetrates more than a quarter or half a millimeter into the lung tissue. Looked at under the microscope the pleura seems elevated or detached from the alveoli, and the blood-corpuscles in the tissue surrounding these alveoli seem squeezed against each other. The subpleural suffusions which accompany pulmonary apoplexy must not be confused with these subpleural spots. They are not supposed to follow death from bronchitis, asthma, or pleurisy, but are associated with "a rapid death which has taken the organism by surprise in a state of normal health or apparently so."

According to Tardieu and other observers, it seems well established that rapid death from phosphorus, arsenic, mercury, lead, and digitalis are likely to be followed by this post-mortem appearance. Dr. Legroux would arrange the cause of these ecchymoses in the same class with that which produces petechiæ of the skin in typhus and typhoid fevers, cholera, purpura, and hemorrhagic variola. These spots may be observed in any case of death which occurs suddenly, in ten or fifteen minutes; MM. Tardieu, Depaul, Tarnier, and other obstetricians have observed them in the fetus which has been exposed by violence to a sudden death, as in pelvic malformations of the mother, or after certain obstetrical operations practised before birth on the fetus, such as craniotomy.[47]

These subpleural spots, therefore, form certain positive signs of an abrupt and sudden death, and are not necessarily confined to any particular poison administered, but rather to the manner of death which has occurred to a person previously in robust health, and unaffected with active disease of any organ of the body.

The ecchymotic spot will not necessarily be found at the location of the injury which produced the contusion; for it is well known that a blow on the frontal bone directly on the brow above the eye will frequently produce an ecchymosis of the lower lid. It is also well known that after a severe blow over the region which covers a soft cavity, like the abdominal wall, ecchymosis is generally observed to occur some time after the blow has been received; but, if a quantity of blood has effused from broken blood vessels in a cavity of this sort, its presence can be determined on palpation of the abdominal wall; if the blow over such a cavity, and even over the thoracic wall, has caused the sudden death of a person, there will in all probability be no ecchy-

[47] See also a thesis of Dr. Dechondeau, Thèse de Paris, 1878, No. 420.

mosis of the skin which covers this cavity. The writer remembers that in the case of his "view" as medical examiner, of the brakeman, which has previously been mentioned in this volume, who was killed by compression of his thorax between the platforms of two railroad cars, there was no ecchymosis of the skin over the region of the heart, though both auricles were ruptured and there was a large effusion of blood and liquor sanguinis in the pericardial sac; the determination of this effusion was easily discovered on palpation. Cabade[48] mentions a case of a robust carpenter who had an attack of epilepsy in his bed. The next day Cabade discovered a mass of black and blue discoloration on his skin over the right side of the trunk of his body; this he attributed to the violent muscular contractions caused by the epileptic fit, because there was not the slightest evidence of any rupture of the heart or of any large blood vessel. It is also well known that the aged and poorly nourished person may have scurvy and purpura, during which, in addition to the small and distinct purpuric spots in the skin, ecchymoses are not unfrequently observed. On the other hand, cases have been noted by Taylor and Stevenson and by Casper, in which violent blows on the chest and abdomen were not followed by skin discoloration, and post-mortem examination of the body showed extensive fractures of the ribs and internal viscera. After severe strains of the tendons of muscles, as also in muscles themselves, black and blue discoloration or ecchymosis is usually observed over the region of these tendons and muscles, which persists for a long time and until the extravasated blood has been absorbed and the blood pigment disappeared.

The case of Christison, recorded in many of the writings on Medical Jurisprudence, warns us that flagellation on the surface of a body of a person just recently dead (within two hours) may produce ecchymosis on the skin which is similar to the ecchymosis caused by contusions from blows inflicted upon the body during life. The period elapsed after death when this deceptive effect can be produced is limited to three hours.

It would be a difficult and useless task in a work of this scope to enter into further detail in regard to the pathological anatomy of the body, and reference is here made to such works as Orth's, which has been frequently referred to among those more recently written, to Ziegler,[49] and to Schmaus and Ewing, as well as to the older writers such as Virchow, Rokitansky, and Sieveking. Where pathological

[48] London Lancet, 1892.

[49] General Pathology, Dr. Ernest Ziegler; Am. translation, Wm. Woods & Co. N. Y. 1900.

appearances are due to a specific poison, these will be detailed under the head of that poison.

In the examination of the stomach it will be found that it presents variable appearances, according as it is inspected during or after the process of digestion, or after long fasting; whether that organ be empty or full, distended or contracted; and whether the distension is due to liquid or to gas.

During and immediately after digestion, the stomach is filled with gas and the remains of the food, and is, therefore, moderately distended, and its mucous membrane appears thin and does not lie in folds. Its color is of a pale rose, uniformly spread over the surface; or, if the organ is unusually distended, it is gray or dirty-white. On the other hand, in the fasting condition it is strongly contracted, and the mucous membrane is corrugated and thick. Its color is of an ashen-gray when it is covered with mucus; but when this is not the case it is of a reddish-brown. It may be partly contracted and partly distended, in which event the differences referred to will be visible at the same time in the pyloric and cardiac portions. Moreover, the cadaveric hypostasis, or settling of blood, will be seen on the folds of the mucous membrane, or in those portions of the organ which are the least distended. After the process of digestion is entirely completed, the abdominal system of veins is loaded with blood, and the same engorgement occurs in certain diseases of the heart and lungs; should death take place at such a time, the mucous membrane of the stomach is found highly injected, and, in consequence of the transudation of liquids taking place in the dead body, ecchymoses are formed which often have the appearance of submucous extravasations; they frequently occupy the entire half of the stomach and both curvatures, and have a bluish-slate color. This injection may also occur in streaks, and thus give rise to an unfounded opinion that death was due to some irritant. This is especially the case where powdered substances, such as arsenic, are found near them, but it is a mere coincidence, since the existence of folds or rugæ is a sufficient explanation of the adhesion of the powder to these places.[50]

The softening of the mucous coat after death is, of course, seen in lesser degrees, and most probably is purely a cadaveric change depending upon the solvent action of the liquids contained in the organ. When the mucous coat is found apparently thickened, this condition is often due merely to the stomach being in a contracted state; and,

[50] Darstellung der Leichenerscheinungen, etc., von Dr. Josef Engel, Professor an der Universität zu Prag-Wien. 1854. 8vo.

on the other hand, it may appear to be very thin when the appearance is solely due to its distention. Similar sources of error to those we have thus cursorily noticed arise in the inspection of the brain, heart, and other organs. The physician should be upon his guard against them, and carefully distinguish the changes produced by disease from those which are brought about in the act of dying, or after death by the position of the body and the transudation of liquids. If familiarity with the ordinary post-mortem appearances does not enable him to form a positive opinion as to the cause of death, it is far better that he should have the candor to say so, than, by giving an unwarranted opinion, incur the risk of causing the innocent to suffer. But in every case it is proper that precise and accurate language should be used in the description of post-mortem appearances, and that such expressions as inflammation, gangrene, etc., which imply the manner in which the morbid change has resulted, should not be used, but rather, instead, such terms as will simply express the physical condition of the part, in reference to its size, color, consistence, etc.

While an extensive discussion on the subject of the general pathology of the organs of the human body should not be presented in a work on toxicology, some synopsis of the pathological conditions usually found in post-mortem examinations of the stomach of a person whose death may have been caused by the ingestion of an irritant poison should be noted. We therefore quote from one of the most recent authorities—Schmaus[51]—a description in general terms of the local effects of the irritant poison on the interior surface of this organ. "The action of poisons on the walls of the stomach is varied, but in general they produce inflammation and necrosis. Some, however, pass into the general circulation without local effect, and others affect the stomach only as part of a general condition.

"The most important local action of poisons is that due to corrosive substances, especially the mineral acids, certain organic acids, and concentrated alkalies. Wherever in the alimentary tract these corrosive matters come in contact with the mucous membrane, they produce an eschar, which may be tough or friable, or loose in texture; and after a time the eschar becomes cast off. In color these eschars differ with the kind of poison; many are stained by the bleeding which they cause; from the destroyed blood they may become brown

[51]A Text-book of Pathology and Pathological Anatomy, Dr. Hans Schmaus; James Ewing, M. D. from the 6th German ed.; Lea Brothers & Co. Phila. and translated by A. E. Thayer, M. D. and N. Y. 1902, p. 360.

or black. The contents of the organ may also be black from this cause. The wall of the stomach may be perforated during life, and frequently is so after death; for the poison continues to act, and may corrode the wall of the stomach and attack neighboring organs post mortem. The factors which modify the corrosion are the degree of concentration, the amount, and the duration of the action. When the poison is taken by accident, the greater portion may be voided from the mouth, and only the upper part of the alimentary tract may present evidences of its action. The amount of material already in the stomach is of great importance; for the acid or the alkali may be partly neutralized thereby, and so diluted as to weaken the action. Metallic salts may form harmless compounds with proteid matters, and rapid use of antidotes and vomiting may prevent or diminish the action of the poison. The effect is most intense when the stomach is empty. The corrosion varies with position, being on the posterior wall of the fundus when the patient lies on the back, and on the fundus and greater curvature in the upright position. When the organ is contracted and the amount of poison small, the edges of the rugæ are the parts to show the action especially. The wall of the duodenum and part of the small intestine may be similarly affected. About the corners of the mouth, corrosive action follows the escape of some fluid during ingestion, or passage of vomit; and the same is true of the lips, the buccal cavity, and the œsophagus. The epiglottis and the aditus laryngis may be corroded and edema of the glottis may result.

"If death is not immediate the stomach suffers severe inflammatory changes, usually of hemorrhagic character. The stomach is strongly contracted, its mucosa or the entire wall is thickened and edematous, and suppuration may follow. The eschars leave deep ulcers when they are cast off, and these may end in perforations. Peptic ulcers may follow later.

"Severe functional disturbances, contraction by scars, stenosis of the cardiac or the pyloric end, hour-glass contraction, and general cicatricial contraction may be some of the later consequences.

"Diluted corrosive fluids have less escharotic action, but the inflammatory effect may be pronounced, and hemorrhagic, or phlegmonous and ulcerative in character. The least degree of such a toxic gastritis resembles simple acute catarrh."

From the foregoing remarks it is, we think, apparent that the most perfect evidence of poisoning is derived from the combined results of the investigation of the symptoms, post-mortem appearances, and

chemical proof. Should any portion of this evidence be wanting, the effect is thereby weakened, but not necessarily always to an equal degree; since a chemical analysis affording positive results, or a decided and characteristic post-mortem change, or a well-marked set of symptoms, may each, in certain cases, afford high probability, if not conclusive demonstration, of the fact of poisoning. On the other hand, the failure of the chemist to discover poison in the dead body does not always destroy the value of other evidence sustaining the fact of its having been taken, since the whole of it may have been removed by vomiting and purging, or, if the patient has lived long enough, it may have been absorbed into the system, and then eliminated from it principally by the urine. The attention of the reader is especially called to the discussion of the detection of arsenic in the urine of persons who have been taking arsenic (in some cases, under careful hospital observation) (§ 12, *supra*) for the purpose of determining how long after the discontinuance of the ingestion of this drug it can be found in the patient's urine. It is now known that arsenic will remain in the system for a much longer period than formerly was supposed.

31. Discovery and demonstration of the poisonous agent, in general.—The intervention of chemistry, and perhaps of physiology likewise, is necessary in this third source of information. If the poisonous agent can be isolated from the tissues of the body, and its poisonous nature be determined to the satisfaction of twelve capable men, then, indeed, have the suspicions assumed an important position in the field of investigation: *Tunc demum res certa erit, ubi venenum ipsum reperietur facile agnoscendum.* Still, this third source of information must not be relied upon to the exclusion of the other two.

In this third source of information, in addition to the chemical analysis, is included also the physiological, physical, and botanical examination. The chemical analysis is not always infallible. There are circumstances which render the detection of a poison by chemical analysis impossible; for instance, the examination may be unavoidably postponed so long as to permit the escape of a volatile poison, like prussic acid, from the body, although it may have been present immediately after death; or life may have been prolonged for a sufficient length of time to permit the elimination of most, if not all, of the poison which was taken, and which was the cause of death, as in the Dr. Alexander case, in which no arsenic could be found in the body, death having taken place in sixteen days after the ingestion of a large dose of the poison.[52] We would refer the reader to Prof.

[52] Medical Times and Gazette, April 18, 1857, p. 389.

Wood's researches on the discovery of arsenic ninety-three days after the last toxicdose, which is mentioned in § 12, *supra*. Nondetection of a poison does not necessarily invalidate the other evidences of poisoning.[53]

On the other hand, traces of poison may be found by chemical analysis when the poison has not been the cause of death; thus, traces of compounds of copper, lead, arsenic, and mercury may be introduced into the body accidentally with the food, or in certain employments, or have been administered as medicine, or in embalming.

32. **Chemical analysis.**— The chemical analysis for the detection of a poison should be complete and exhaustive. The poison should be isolated and so identified as to admit of no possible doubt. Fortunately, the progress of organic chemistry during the past few years has been so great that the chemist is now able to isolate and identify many of the organic poisons with as much certainty as the metallic poisons; thus he can isolate, so as to obtain in a pure crystalline form, weigh and test chemically, microscopically, and physiologically, morphin, strychnin, and many of the other organic poisons, as readily as he can arsenic, mercury, or any of the metals.

The chemist must, in legal analyses, take precautions similar in character to those recommended above for the physician in making the autopsy and preserving the identity of the organs. All of his work must be performed by himself; his apparatus must be perfectly clean, and must have been previously carefully tested; his chemicals must also be of known purity, by having been previously thoroughly tested; while an analysis is being performed, all of his materials, viscera, and other substances to be analyzed, chemicals, and apparatus must be kept under lock and key, so as to prevent the possibility of being tampered with. Since, in the analysis for many of the poisons, the material removed from the body is to be destroyed by chemical reagents beyond the possibility of restoration, this material should be first subjected to a thorough and systematic preliminary examination for the purpose of obtaining some hint as to the nature of the poison present, and to prevent the waste of material by being obliged to search unnecessarily for poisons not present. On this account also the chemist should be furnished with all the information which it is possible to gain in any given case from the symptoms, pathological

[53] This has been recognized in legal cases in Prussia. Vierteljahrsschrift f. gericht, Med. 1857, XXXII. pp. 177 and p. 96. 193; 1858, XIV. p. 185; 1860, XVII. p. 177; 1862, XXI. p. 1; 1864, N. F. I.

appearances, and circumstances; since, if his suspicion be strongly directed to any particular poison, he may proceed at once to test for it without wasting any of his material (which is always very limited in amount) and thus losing valuable time in useless work. This preliminary examination should include a careful and systematic examination of the mucous membrane and contents of the stomach and intestines, with the naked eye, with an ordinary magnifying lens, and, if anything suspicious be seen, such as any white or yellow patches, it should be examined with a microscope. The odor should be carefully noticed as well by the chemist as by the physician. The contents of the stomach should always be examined under the microscope, since, frequently, undissolved portions of the poison, such as arsenic, may be seen, or the character of the food which was last eaten by the deceased, and the amount of digestion which it has undergone, may thus be determined, thereby, perhaps, throwing light upon the time of death in uncertain cases, or giving other information, the importance of which may not be apparent at the time of the examination, but may prove of vital importance at the subsequent inquest or trial. The chemist, in addition to the material removed from the body, may be called upon to analyze and examine a large number of substances, such as food suspected to be mixed with the poison, stains upon clothing, bedding, carpets, or even portions of the floors where vomitus has fallen, any paper or bottle containing an unknown substance found in the neighborhood of deceased, or among the effects of any suspected person, etc., etc. If by such preliminary examinations he can obtain any hint as to the probable nature of the poison which was the cause of death, he should proceed at once by the most direct methods to test the viscera for that poison, but, if no such hints can be obtained, he must resort to the systematic methods of analysis for the isolation of poisons from organic substances. These methods are various, and need not be mentioned in detail here; they are given in special works of chemistry, and are familiar to expert chemists.

When the nature of a poison has been determined by analysis, it is, of course, important to ascertain the amount of the poison present; this is not, however, of vital importance; that is, if a fatal dose cannot be isolated from the body, it does not show that a fatal dose had not been taken before death, because the whole of a body is never submitted to analysis, and a portion of the poison remains in those parts of the body not examined, and if a person lives sufficiently long after taking a fatal dose of poison, a portion or even the whole of the

poison may be eliminated before death, and yet the death have been due to the direct action of the poison.

Finally, the nature of the poison must be determined with positive certainty. It must be isolated in a form so pure that it can be recognized by all of its properties, physical, physiological, and chemical. If a metallic compound, the metal itself should always be obtained, and if an organic poison, the crystals, if it be crystalline, should be obtained. With regard to the possibility of poison being surreptitiously introduced into the stomach after death with a view of casting suspicion upon others, we may safely say that its consideration is not required until some authentic instance of the fact can be produced. This is one of the chimeras of medical jurisprudence, which the ingenuity of authors has evoked, but whose existence is fabulous, if not absurd.

33. Differential diagnosis of poisoning.— The physician is often at a loss, upon the first view of a case of poisoning, to determine whether the symptoms presented by the patient may not be really due to disease. The aspect of a case of irritant poisoning presents a certain resemblance to cholera or to gastroenteric disturbance, and most of the phenomena of narcotic poisoning are found in diseases affecting the brain or spinal cord. Should he content himself with remaining a silent spectator of the case, he may remain in doubt until its close; but if, on the contrary, he has witnessed or been made aware of the time and circumstances under which the symptoms came on, and their mode of invasion, he will be less embarrassed, and, if an autopsy is obtained, he can seldom be at a loss to give a decided opinion. The diagnosis must remain incomplete without a post-mortem examination, whether its results be positive or negative, unless we except certain cases in which a chemical analysis of the excreta has clearly revealed the presence of a poisonous agent, in which case the combination of symptoms and chemical detection of the poison make an important link in the evidence; and hence, for practical ends, the careful analysis of symptoms, in those diseases which leave but few traces behind them, is of primary importance.

Reliance upon symptoms alone, no matter how suspicious the suddenness of the attack may be, must never, to the mind of a professional expert, be a presumption of criminal poisoning; because it is well known that many diseases are marked by the sudden appearance of symptoms which are almost directly followed by death. This prejudgment by symptoms alone may not only do a lifelong injury to an innocent person, but may even defeat the ends of justice, by pre-

venting a cautious and quiet investigation of the cause of a suspicious death.

We have, in the foregoing pages, cautioned the medical practitioner that there is an instance on record where the medical attendant was misled by positive symptoms of disease, which occurred simultaneously with the effects of a poisonous drug, to sign a certificate of death as caused by the disease; whereas, it was proved in court that a murderess was all the time present in the house, with probable intent to murder the victim; moreover, this same murderess was convicted of the murder of another member of the family, and in the same house, for the motive of securing the benefits of a life insurance policy. There were five persons' lives which would have prevented her from securing the benefits of this policy; their deaths were subject to suspicious circumstances which were fully brought out at her trial for murder. We would refer our readers to the particular comments on this trial of Sarah Jane Robinson as given on a previous page (p. 31). It only concerns our present purpose to note the fact that the medical attendant upon one of the supposed victims signed, as the cause of her death, a certificate which stated that she died from pneumonia, but, afterwards at the trial, stated that in his opinion she had probably been the victim of a poisoning by arsenic.

It must not be forgotten that pathological conditions, preceded by symptoms, may cause confusion, in simulating certain acute as well as chronic diseases, and sometimes even the post-mortem results of these diseases. For an instance of this, reference is made[54] to a case of poisoning by arsenic, resulting in a general fatty degeneration of the organs, which might have formerly passed under the head of a death from natural disease. This case, however, is one of the many which help to establish "the fact that arsenic, in common with phosphorus, antimony, and other poisons, will cause fatty degeneration of the liver, kidneys, gastric glands, heart, and other organs. . . . Fatty degeneration is a frequent result of arsenical poisoning. Probably either it or the preceding condition of granular degeneration always exists."

On the other hand, "slow poisoning," or the result of the repeated administration of nonfatal doses of a poison, may, by its symptoms, simulate the appearance of a disease. In these cases, if suspicion has been directed towards an unnatural cause of death, chemical investigation and a careful post-mortem examination should form con-

[54] Transaction of the Massachusetts ton, 1878; and in Boston Med. and Surg. Medico-Legal Society, Vol. I. p. 43, Bos- Jour. Sept. 19, 1878, p. 357.

elusive testimony. Again, the coincidence of disease may suggest to a criminal a means of diverting suspicion, and we may have a case presenting either a conflict of opposing symptoms due to the effects of a disease and a drug, or a confusion of similar symptoms of both. A case of this character is represented by the trial at Nashua, New Hampshire, of E. W. Major for the murder of his wife by strychnin in December, 1874.[55] The strychnin was given to her while in labor, and the convulsions occurring were at first attributed to the effect of the patient's condition. The discovery of strychnin proved the fallacy of this opinion; and in fact it may safely be said that the convulsions occurring in puerperal eclampsia (which are most probably a symptom of functional or organic disease of the kidneys resulting in uremic poisoning) may be numerous, whilst the convulsions due to strychnin poisoning are rarely more than five before the fatal result of poisoning occurs; and during the interval between the convulsions in strychnin poisoning the patient is conscious, while there is unconsciousness for a long time after a puerperal convulsion.

34. Diseases liable to be confounded with symptoms of poisoning, in general.— For convenience we may consider the various diseases which are liable to be confounded with the symptoms of poisoning, under the same heads in which the various poisons are classified by some toxicologists.

1st. Irritant; *viz.,* cholera; cholera morbus; gastritis or inflammation of stomach; enteritis or inflammation of smaller bowel; colitis or colic, usually the result of a fecal accumulation in the larger bowel, and rarely fatal when properly treated; appendicitis, often resulting in peritonitis or inflammation of the serous membrane which lines the whole interior of the abdominal cavity and most of the abdominal organs; dysentery; and typhoid fever.

2d. Hypostheniants; *viz.,* pneumonia; puerperal and uremic convulsions; epilepsy; and tetanus.

3d. Stupefacients; viz., certain diseases of the brain; apoplexy; septicemia or blood poisoning; embolism or obstruction of arteries by clots; typhoid fever; and dysentery.

4th. Narcotics, uremia, diabetic coma, and certain diseases of the brain marked by the state of coma, pneumonia, and typhus or ship fever.

5th. Neurosthenic or neurotic poisoning; *viz.,* cerebro-spinal meningitis, cerebral abscess, and certain acute diseases of the brain

[55] See Appendix.

marked by delirium; and many other obscure diseases of the nerve centers.

It would be impossible to enumerate all the forms of disease which might be mistaken for symptoms due to poisoning, for it is not that diseases are insufficiently marked by symptoms, but because these latter, in the present system of medicine, are referred to well-known pathological conditions or lesions of limited and definite organs concerned in performing the functions of life. Poisons, on the other hand, generally act upon more than one organ at the same time, and without necessarily causing the same conditions or lesions of organs which are associated with the symptoms due to these lesions; for instance, in pneumonia, life is destroyed by reason of a definite and well-known unhealthy condition of the lung substance interfering with the function of breathing, while hydrocyanic acid, which may also cause death by signs of asphyxia, does not cause a similar lesion of the lung substance.

The diseases most apt to be mistaken for irritant poisoning are:—

35. Cholera.— This disease in its malignant form, as is well known, is often rapidly fatal; it may supervene shortly after a meal or a draught of liquid; its onset, marked by a lowered bodily temperature, is sometimes sudden, although usually preceded by diarrhea; there are great thirst, vomiting, and purging, without effort, of a thin and slightly turbid liquid; the surface is cold and shriveled, the features collapsed, the voice almost extinct, the pulse feeble or imperceptible, and the intellect undisturbed; the lesions discovered after death are not sufficiently characteristic to be used in evidence; sometimes diphtheritic changes are found in the intestines, sometimes a deep injection of their submucous coat. In most of the symptoms enumerated, cholera may resemble very closely a case of poisoning by arsenic or other irritant, but there are, nevertheless, sufficient means of distinction.

"The post-mortem appearances of Asiatic cholera vary with the stage of the disease. In the early stage (the algid) the intestine is filled with a whitish fluid, the 'rice water' discharge, which is odorless and made up of serum and flocculi of mucus, and in it comma bacilli are to be found in countless numbers. The mucosa of the ileum is very red and swollen, at first covered with a layer of thick mucus. Few or many small foci of hemorrhage may be present, and the follicles are swollen and gray or reddish. Rupture of these may make small ulcers. The colon is less affected or not involved. Mi-

croscopically, there are the lesions of severe catarrh and profuse infiltration.

"After the algid stage a period of general infection may follow, called the cholera typhoid; and, if death occurs now, the intestinal condition may be negative. The hyperemia has disappeared; the mucosa is pale or marked by blood pigment; its contents are bile-stained, and not so fluid; and in the colon masses of firm feces may be found. In many cases a diphtheritic sloughing of the mucosa may take place, with large, deep ulcers and hemorrhages.

"In general the blood is thickened, because of the great loss of fluid by the intestine, dark and imperfectly clotted; the right ventricle and large veins filled with blood, and the left empty. The serous membranes feel slimy and are covered by a thick, albuminous, sticky layer. The spleen is not enlarged. The kidneys present cloudy swelling, fatty degeneration, or acute nephritis, and, in some cases of cholera-typhoid, nephritis and uremia are the chief factors. The liver is in parenchymatous degeneration; the cadaver is cyanotic; the muscles, especially of the calf, are rigid; rigor mortis occurs early and persists, and the abdomen may be much retracted.

"The accepted cause of cholera is the comma bacillus. In doubtful cases its discovery may be the only satisfactory proof of the diagnosis; for the severer cases of cholera nostras are so nearly identical in symptoms with the Asiatic type that only the presence of the vibrio can determine the disease. Both microscopic examination of the stools and cultivation must be employed. The cause of cholera nostras is unknown." [56]

In poisoning by the irritants, a burning sensation in the throat and stomach, and pain and distress in the whole abdomen, but chiefly over the stomach, precede, or occur simultaneously with, the vomiting. These are the most prominent and constant symptoms during the continuance of the case. The matters passed from the stomach and bowels, after their previous contents have been evacuated, are mucous and bloody, and are not spouted forth as in cholera, but rejected with great distress and effort. The anus is often, indeed, excoriated by their irritating properties. Furthermore, it may be observed that those who have once witnessed a case of malignant cholera will most probably have the peculiar but indescribable features of this disease so impressed upon their memory that they will not readily mistake for it any case of irritant poisoning. Finally, the epidemic prevalence of the disease, or the fact that about the same time other cases

[56] Schmaus and Ewing, op. cit. p. 372.

resembling Asiatic cholera have occurred, will materially assist the physician in giving a positive opinion as to the nature of the attack.

36. Cholera nostras, or cholera morbus.—Cholera nostras, or cholera morbus as it is usually termed, is a disease which has more points of resemblance to the effects of poisoning than that which has just been mentioned. In it, both the extreme collapse and the peculiar rice-water discharges are not seen; but, on the other hand, the vomiting and purging are of a bilious character, and there is excessive pain in the abdomen. The progress of the case is, however, different. The pain in cholera morbus is remittent, coming on in paroxysms; and in proportion as the offending matters are discharged, the vomiting is less frequent and painful. In irritant poisoning, on the contrary, the pain is constant, and there is usually also tenderness upon pressure; the vomiting is of mucus and blood, and the discharges from the bowels are of a similar character. The tendency in the latter is to death, in the former to recovery. Cholera morbus is seldom fatal, and when it is, death does not, in general, take place for several days. The contrary is the rule in poisoning by the irritants. Such are the distinctions usually advanced by authors; and while they are, as a general rule, undoubtedly correct, it should not be forgotten that distinctions valid in medicine may not be so in their application to criminal cases. While, in the science of medicine, diagnosis is founded upon a careful investigation of the prevailing and general characters of diseases, the most delicate questions in medical jurisprudence are, on the other hand, determined by exceptional cases. Now, as experience shows that persons may not die from the effect of the poisonous irritants until several days have elapsed, or, indeed, that they may not die at all, we are at a loss to perceive how, in such cases, it will be possible, in the absence of circumstantial and direct evidence, to decide that the symptoms were due to attempted poisoning, rather than to the disease in question; for, although there may be some points of distinction, as, for example, the early occurrence of a burning sensation in the throat, the unremitting character of the pain, and the sanguinolent discharges, yet these may be absent in mild cases of poisoning, and where life is not destroyed.

It may be observed, as a further aid in differential diagnosis, that diarrhea in the earlier stage of cholera morbus will be usually absent, and sometimes does not occur at all, while in the case of irritant poisoning this is an early symptom.

The corrosive poisons leave traces behind them sufficiently distinct

to prevent any likelihood of mistaking their effects for those of disease.

37. **Rupture and perforation of stomach.**— There are certain diseases or sudden accidents partially resembling in their symptoms those of the irritant poisons, which are so readily recognized on post-mortem examination, that it is needless to enlarge upon the modes of distinguishing them. These are rupture of the stomach, intestine, biliary ducts, and uterus, and no one will contend that such lesions can be produced by poisoning.

The post-mortem rupture of the stomach must be carefully distinguished from that occurring during life, and will be accompanied by the escape of a varying amount of the contents of the stomach into the abdominal cavity, and an absence of inflammatory action. "The walls of the stomach, especially at the fundus, (or lower portion) are much softened, and are frequently converted into a translucent, slimy mass."[57] If the digested condition of the stomach walls which surround the opening be due to the action of a corrosive, as, for instance, sulphuric acid, there can hardly fail to be present certain inflammatory signs due to its more general action upon the internal surface. "The effect produced by perforations occurring during life ranges according to the relations of the surrounding parts; a communication may be established with the abdominal cavity, or with a closed sack, resulting from chronic inflammation, or the hole in the wall may become closed by the formation of adhesions between the stomach and other organs. In the first instance, the result is a general peritonitis which is quickly fatal; in the second, a circumscribed purulent or ichorous inflammation, which is frequently of long standing; and in the last, as a rule, a chronic progressive ulceration. In such cases it is frequently necessary to vary from the usual method of performing the autopsy, and to remove the stomach in connection with the other organs, most frequently the pancreas and liver." [58]

The symptoms which precede death by perforation of the stomach or intestines deserve but a passing notice. These may, however, give rise to embarrassment, not, indeed, so much from the symptoms of the disease when considered alone, but from their offering, perhaps, sufficient resemblance to those of poisoning to support the opinion that the lesion referred to may be due to this cause. While it is true that in this disease the seizure is sudden and the pain in the abdomen

[57] Orth, Diagnosis in Pathological Anatomy, translation, N. Y. p. 281.

[58] *Ibid.*

acute, it is not preceded by the sensation of burning in the throat and stomach, nor is the vomiting urgent unless upon the ingestion of the liquids. There is, again, no diarrhea; the main symptoms are acute, diffused pain all over the abdomen, arising from peritonitis, and the patient is collapsed from the first. But in a case of this kind, which has not been closely observed, the discovery after death of the perforation in the stomach will naturally awaken the suspicion of poisoning. If we now inquire under what circumstances this lesion is produced in cases of poisoning, we will find that with the exception of the corrosives it is seldom occasioned by any kind of poison. Perforation from arsenic is so rare an event that but three cases are said to be on record, and the fact of the perforation being so unusual in a form of poisoning so exceedingly common, renders it highly probable that in these instances it was due to an already diseased state of the coats of the stomach. But the corrosive poisons, which undoubtedly produce, in many instances, a perforation of the stomach, leave in addition such manifest traces of their action upon the throat, œsophagus, and stomach, not to mention the corrosion of the mouth and lips, that it seems to be inexplicable how the single fact of the perforation should leave any doubt in the mind of the examiner or of the jury. Moreover, the character of the perforation alone affords a sufficient ground of distinction. The stomach in such cases is blackened and extensively destroyed; the aperture is large, its edges rough and irregular, and the coats are easily lacerated. Further, the poison escapes into the cavity of the abdomen, where it may be easily discovered by chemical analysis. In perforation from disease, on the other hand, if the affection be of a cancerous nature, there will be no difficulty in distinguishing the cause, and, if it be simple ulceration, such as occurs sometimes in persons in the enjoyment of apparently good health, the opening is also characteristic in its nature. "The aperture is usually of an oval or rounded form, about half an inch in diameter, situated in or near the lesser curvature of the stomach, and the edges are smooth. Indeed, it has not unfrequently the appearance of having been 'punched out.' The outer margin of the aperture is often blackened, and the aperture itself is funnel-shaped from within outwards: *i. e.*, the mucous coat is the most removed, and the outer or peritoneal coat the least. The coats of the stomach, round the edge of the aperture, are usually thickened for some distance; and when cut they have almost a cartilaginous hardness." (Taylor.) Death takes place from peritonitis, the contents of the stomach escaping into the cavity of the abdomen.

Similar perforations may exist in the duodenum or upper portion of the small intestine, but they are comparatively rare. Where these perforations of disease occur, they are usually the result of the round or chronic ulcer, or more rarely still of cancerous disease. All these perforations resulting from disease are quite small, the diameter rarely exceeding 1/10 of an inch.

There is still another form of destruction of the walls of the stomach, which is due to a maceration of the stomach by its contents after death. It cannot give rise to a suspicion of poisoning, unless in the absence of any knowledge of the symptoms preceding death. It is purely a cadaveric phenomenon, and may occur in the stomach of persons dying from any cause, provided the peculiar digestive process necessary for its production exist.[59] It is formed only in the larger end of the stomach. The opening is large and irregular, with ragged and "moth-eaten" edges and no surrounding inflammation. The edges may be discolored and black, as the result of a chemical action of the intestinal gases upon the coloring matter of the blood. There is no peritoneal inflammation; but the spleen, diaphragm, or other subjacent viscus may be softened by the acid. The absence of any disorganization of the pharynx and œsophagus, and of peritoneal inflammation, is sufficient to distinguish this post-mortem perforation from that caused by corrosive poison, with which alone it is possible to confound it. Dr. Budd has found, naturally enough, that this post-mortem softening of the coats of the stomach is more common in hot weather. He says: "During the past summer, which was a very hot one, my attention was casually drawn to this subject, and from the middle of May to the middle of August I carefully examined the stomach in all the bodies that were opened in the King's College Hospital. In several instances the mucous membrane of the stomach, in the greater curvature, was completely destroyed, and in a very large proportion it had been clearly acted upon more or less by the gastric juice. I renewed my observations in October, but the change, in a striking degree at least, was then much less frequent." [60]

Should the rules thus laid down for the discrimination of the source

[59] See Medical Times and Gazette (No. 246, p. 268) for a case in which the stomach of a child (which had been asphyxiated by its intoxicated mother hugging it too closely) presented the following appearances: Nearly the whole of the great cul-de-sac had disappeared; the edges of the aperture were thin, jagged, and flocculent; another similar but smaller opening existed lower down, and abutted upon a corresponding aperture in the transverse colon. No unnatural adhesions or other morbid appearances existed, and a quantity of milk was found in the stomach and in the cavity of the abdomen.

[60] Lancet, 1847, p. 593.

of these perforations not prove sufficient, a resort to chemical analysis will render the demonstration complete. If any poison has been taken in so large a dose, or is possessed of such violent properties as to cause the lesion thus referred to, it will readily be found by these means. It has been said that a person may die with the symptoms of irritant poisoning, and after death, perforation, the result of cadaveric change, be found, and that hence the knowledge of the true cause of the perforation does not exclude the idea of poisoning. It is true that such a rare coincidence may happen, but the want of connection between the poison and the perforation merely renders it necessary to support the charge upon other evidence. The case of Miss Burns, for the murder of whom, by poison, a Mr. Angus, of Liverpool, was tried in 1808, is one in which this doubt arose. The charge of poisoning was not sustained by chemical or pathological evidence, and the prisoner was acquitted.

38. Gastritis, gastroenteritis, peritonitis.— It is the natural effect of poisonous irritants to produce one or more of these diseases, but as they may arise from other causes, a distinction is in practice necessary. Gastritis has a more protracted course than is usual in irritant poisoning. Diarrhea, so nearly universal a symptom of irritant poisoning, is not always present in these diseases. These names signify inflammation of stomach, or of intestines, or of the peritoneal surface of the abdomen, which, like other inflammatory processes, is always accompanied with fever or an increase of bodily temperature; hence, the natural effects of disease may be simulated, but if the medical attendant be a careful observer, the difference between the symptoms of an irritant poison, which may induce secondary inflammatory processes, and a natural disease, will hardly escape notice. However satisfactory these distinctions may be to a physician, it is apparent that they may have little weight with others; hence, practically, it is important to examine them closely, for the accusation in such cases will have to depend upon the results of the chemical investigation.

39. Strangulation of intestines.— Strangulation of the intestines has been enumerated among the diseases likely to awaken suspicion of poisoning, but with little justice; for if the symptoms are not sufficient to distinguish it, most certainly it cannot fail to be detected upon the post-mortem examination.

40. Diseases of brain, in general.— The symptoms produced by narcotic poisoning may be closely imitated by those of natural diseases, such as apoplexy, epilepsy, congestion of the brain. Until

recent years tetanus, as a disease, could not be recognized by its post-mortem effects as distinct from death by certain poisons; now, however, Schmaus, among other pathologists, calls attention to its recognition, by the presence at the seat of external injury of the bacillus tetani. This bacillus has its origin in garden earth, hay-dust, on splinters of wood, in decaying fluids, and the feces of horses and cattle. "It is a straight rod, bristle-like, which makes end spores, and with its swollen end resembles a drumstick or large-headed pin. It is motile and anaerobic." These bacilli are found in the tissues only at the point of entrance in the wound through which they have been introduced. "They produce toxins by which the general intoxication is caused. So-called rheumatic tetanus (without discoverable wound) is probably due to infection through the lungs." In regard to narcotic poisons and the confusion from symptomatic similarity of above-named and natural diseases without post-mortem signs, occasionally, the similarity is so great that, upon the medical evidence alone, it may be impossible to acquire a certainty of the cause of death. Many distinctions have been drawn by writers upon toxicology between the effects of narcotic poisons and those of disease of the brain and spinal marrow, but they serve only to show the very close analogy between them. When, moreover, it is remembered that the most important of these poisonous agents leave no distinct traces of their action in the dead body, it will be perceived that the differential diagnosis must depend mainly upon the results of chemical investigation, and the direct or circumstantial evidence in the case.

The use of instruments, for instance, in discovering by the stethoscope a condition of the lungs often associated with cerebral or brain diseases, the thermometer to indicate inflammatory processes due to disease of the nerve center, and other means now more familiar to the medical profession than formerly, may serve to differentiate disease from acute poisoning. An effusion upon the membranes covering the brain causes a very rapid appearance of lowered bodily temperature, followed suddenly by increase of temperature; the action of opium tends to a depressed bodily temperature, yet narcotic symptoms are present in both cases. Unfortunately, no carefully recorded observations of bodily temperature as the result of acute drug poisoning have been presented in courts of law; yet, we believe it would prove of material value if a point of this kind were insisted upon in medical testimony in cases where reliance is placed upon symptoms. The thermometry of disease is pretty well understood by the

educated physician, and the experience of every additional year tends to make our knowledge more exact.

41. Apoplexy.—Apoplexy, it is said, may be distinguished from opium poisoning by the following considerations: That it does not usually occur under the age of thirty, nor come on without warning symptoms, and that the time of seizure is irrespective of the taking of food or drink. But those distinctions are futile, even when, as is rarely the case, an accurate account of the whole history of the sickness can be obtained. More reliable are the facts that, in poisoning by opium, the symptoms are gradual in their accession, and that the more confirmed effects are preceded by drowsiness, and that the patient, until an advanced period of the stupor, can be temporarily aroused. The pupils also, in general, are strongly contracted, and there is no contortion of the face or paralysis of the limbs. Dr. Luff refers[61] to the fact that "hemorrhage into the pons varolii has been mistaken for opium poisoning, on account of the conjunction of insensibility with contracted pupils." In the majority of fatal cases of apoplexy the attack is sudden, although indistinct warning symptoms may have preceded it; the patient cannot be roused, the pupils are dilated and insensible, and the face is slightly contorted, indicating a paralytic condition of one side of the body. Yet there are numerous exceptions to these rules, a fact which is easily understood, when we reflect that opium in addition to its specific narcotic properties produces the very same pathological condition, with the exception of effusion of blood into the substance of the brain, from which it is our aim to distinguish it; this need not, however, cause confusion, for chemical science can easily determine the presence of opium or morphin in the brain substance in those cases in which its use has been followed by death, when it has not been destroyed or eliminated from the organism.

42. Embolism.—A clot formed in the heart or larger blood vessels may be sent into the arterial circulation, and plug up an artery which feeds an important organ,—the lungs, for instance,—and, without manifest symptom, may cause sudden death. This is not so unusual a cause of sudden death as may be generally supposed; and, as the person may have been previously in robust health, this catastrophe, not unfrequently occurring in bed, may give rise to suspicion of foul play. This accident may also occur in any febrile attack or during convalescence of a lying-in patient. A careless autopsy may have

[61] Op. cit. Vol. I. p. 260.

caused the pathological proof of an embolism to be overlooked; because often these clots are quite small, or fall out by rough cutting or handling of the blood vessels.

43. Pneumonia.—Death from edema (dropsy) of the brain or lung substance, from sudden shock, or from carbon monoxid poisoning, may supervene so suddenly that the individual may die before the arrival of a medical attendant. In fact, cases are recorded where the first sign of pneumonia was a sudden fall in the street, unconsciousness, coma, and death following almost immediately. Here, again, the post-mortem examination reveals the natural cause of death.

44. Cerebro-spinal meningitis.—Symptoms of this disease were confounded with those of poisoning by tartar emetic, in the trial of Mrs. E. G. Wharton, at Annapolis, Maryland, in January, 1872. Without attempting either a review of that case,[62] or a thorough description of the above disease, it may be advisable to relate the character of the symptoms most usually noticed in this serious epidemic disease. Dr. Ed. Warren, in the case[63] alluded to, stated that the patients are in a "condition of semiunconsciousness and of increased sensitiveness of the surface of the body, so that they shiver when touched; and have a rigidity of the muscles of the neck, back, and inferior extremities; pupils neither contracted nor dilated, but insensitive to light; suppression and retention of urine; lividity of countenance; trismus; opisthotonos; occasional jactitation and restlessness; incoherent articulation, and a speedy and violent death."

This disease occurs most generally as an epidemic, and is more frequent in winter and spring than in the summer months. The best authorities (Niemeyer, Aitken, Valleix, and in this country, J. B. Upham, A. Stillé, W. H. Draper, S. Ames, and others) describe the disease as commencing, often with little or no positive warning,[64] with a severe chill of variable duration, accompanied by a severe headache, and in most cases by vomiting. At the end of the first or second day, rarely later, the head is drawn backward. The severe headache continues, the pain extending down to the nape of the neck and along the spine. The third or fourth day the muscular contraction, especially in the vicinity of the neck and back, is quite remarkable, and to this succeeds opisthotonos (or an arching of the

[62] Am. Jour. of Med. Sci. April, 1872, pp. 329 *et seq.*

[63] Reported and published by the Baltimore Gazette, p. 97.

[64] A police officer at Dublin, Ireland, was stricken down suddenly while on his beat, became unconscious, then had violent convulsions, and was dead in less than ten hours, though he had, the same morning, left his house apparently in good health.

back in such a way that the body may rest only upon the heels and back of the head); consciousness is lost; constipation, rarely diarrhea, continues throughout the whole progress of the disease; the pulse is not generally more rapid than in health, though it may be irregular and soft; towards the close of the disease, if fatal, the pulse is more rapid. The urine is suppressed or retained. The peculiarity of the respiration is a marked feature of the disease.

The post-mortem appearances of this disease are more marked if the disease has existed several days before death. If the patient dies during the first stage, *viz.*, before exudation occurs, the appearance of the brain and spinal cord is not peculiar, and oftentimes no great change in any organ has been remarked.

Usually the appearance, as described by Niemeyer, in recent cases is as follows: "The subjects show no emaciation, protracted rigor mortis, or extensive hypostasis. The cranium contains much blood; the dura mater is more or less dense and occasionally covered with small hemorrhagic spots. There is usually no effusion between the outer membranes; in the subarachnoid space there is an exudation, which, both in extent and character, occupies about a medium position between the purulent exudation in meningitis of the convexity, and the puro-serous exudation in meningitis of the base of the brain." The brain itself is more or less vascular. The dura mater of the spine is also more or less vascular, occasionally very tense, especially at the lower part. The arachnoid usually presents no peculiarity except a decided opacity. The spinal medulla itself is more or less vascular, occasionally infiltrated and relaxed. Schmaus and Ewing state that "in many cases of purulent cerebro-spinal meningitis the lanceolatus streptococcus is found, in others, the streptococcus pyogenes, and in others, a form nearly related to the first, enclosed within the cell body of the nucleus of cells." Dr. Ernst Ziegler[65] states that "Jäger is of the opinion that the diplococcus intracellularis meningitidis is the cause of epidemic cerebro-spinal meningitis, and is entirely distinct from the pneumococcus. Sporadic meningitis may, on the other hand, be caused also by the pneumococcus." If these theories are substantiated the bacteriologist could settle a doubt.

Except some accidental complications, there are no particular anomalies of the other organs; we should especially note here that the spleen is almost always normal.

[65] General Pathology, Dr. Ernest Ziegler. Translated under Editor Dr. Albert H. Buck; William Wood & Co., New York, 1900, p. 461.

45. Tetanus.— The same remark is applicable to the resemblance between the convulsions of tetanus and those produced by strychnin. It is needless, therefore, for us to draw a parallel between the diseases referred to and the symptoms so closely imitating them, produced by the poisonous narcotics. Where the success of either prosecution or defense comes to stand upon such vacillating ground as this, other sources of evidence failing, it would be better that the most approved works on pathology should be consulted and the descriptions of disease there given be compared with the symptoms enumerated under the narcotic poisons, than that the reader should draw an unwarranted conclusion from such an imperfect abstract as it would be proper for us to give in this place.

In Palmer's case the distinction between the symptoms of poisoning by strychnin, and tetanus, was thoroughly investigated.[66]

Finally, all cases of sudden death may awaken suspicion of poisoning. If, as may well be the case, the post-mortem alterations do not clearly indicate the seat and nature of the affection, a review of the manner of dying, and the absence of any positive result from chemical investigation, must at once negative the presumption of poison having been taken. Thus, certain diseases of the heart, overdistention of the stomach, fatal syncope, and some obscure diseases, may not be recognized at the autopsy, but the mode of death in them is entirely different from that in any form of poisoning, except, perhaps, by prussic acid, in which the odor or chemical tests will disclose the cause of death. The importance of a careful collection and comparison of all the medical evidence in every case cannot be too strongly insisted upon; for upon this combination of proof it is that a correct knowledge of the true cause of death must depend.

46. Compound poisoning; complication of symptoms due to the combined action of poisons.— The so-called antagonism of medicines is certain to play an important part in the subject of toxicology, and deserves mention here. By antagonism of medicines is meant the results of observation of symptoms of one medicine as opposed by the symptoms of another, or the modification of one set of symptoms due to a poison by those of an entirely antagonistic character; for instance, the dull, heavy narcotism induced by an overdose of morphin may be antagonized by a moderate dose of atropin. In the former class of symptoms we have a slow respiration, say ten to fifteen inspiratory movements of the chest in a minute, and a slow

[66] London Lancet, May and June, 1856.

pulse, forty or fifty beats per minute; whereas atropin quickens both respiration and pulse; and both of these drugs will produce a mental hebetude, and even coma; though atropin, in moderate doses, will induce an active delirium which is gay and lively, while opium produces a passive delirium which is dreamy and lascivious. Again, the quieting influences of chloral, which may obtain until death occurs, may be antagonized by the convulsifying action of strychnin. Antagonism of medicines does not necessarily imply that the poisonous dose of one drug may control the issues of life and death set in danger by another drug; for instance, a nonfatal dose of strychnin may antagonize the symptoms induced by a fatal dose of chloral, provided the stychnin be first given; while, again, a nonfatal dose of strychnin given to a patient whose life is already jeopardized by a poisonous dose of chloral may even accelerate the fatal issue. Still further, the complication of symptoms may be masked by the use of a third or fourth drug. An illustration is offered in the following cases:

Dr. Harrison reports[67] the case of an attempted suicide by the combination of strychnin and opium. About ¾ of a grain of strychnin and ninety drops of laudanum were swallowed, and in three quarters of an hour the strychnin convulsions appeared; during this time some urine, though only a portion of that passed was collected from the man, showed the presence of 1/20 of a grain of strychnin. Chloral was used as an antidote to the strychnin; the man recovered. Another case is reported,[68] where a similar amount of strychnin was swallowed combined with 2 drams of laudanum and some oxid of mercury. No convulsions occurred, but the man exhibited an intense narcotism which gradually disappeared. Dr. Marvin[69] reports the case of a patient who took, with suicidal intent, 10 grains each of strychnin and morphin during an attack of melancholy, which a post-mortem examination proved to be due to cerebral condition caused by uremic poisoning from serious disease of the kidneys. The symptoms of morphin poisoning could hardly be recognized.

The second case reported by Dr. Marvin[70] was seen by him in consultation, and was remarkable from the fact that the antagonism was caused by disease; in this case serious coma and apparent narcotism supervened after the moderate ingestion of 1¼ grains of morphin administered in fractional doses of ¼ of a grain every three hours. Coma with stertorous breathing occurred four hours after the

[67] Lancet, May 13, 1882, p. 780.
[68] Lancet, Dec. 23, 1871.
[69] Med. Herald, Louisville, 1879, No. 1.
[70] *Ibid.*

final dose, or fifteen hours after the first dose was taken; an hour afterwards 1/60 of a grain of atropin was administered subcutaneously, because the respiratory movements were slow and feeble, and the pulse weak, with a bodily temperature of 103° and 106°. To this was added the stimulating effect of electrical treatment. The cerebral symptoms were so marked, and the patient so nearly dead, that Dr. Marvin left him, as he supposed, *in articulo mortis;* but he learned from the attending physician the next morning that the patient recovered from these severe symptoms. An examination of the urine revealed a low specific gravity, one third of albumin, and a number of waxy and granular casts. The high temperature would show that the cerebral lesions were not due to uremia, but that this condition made the narcotic effects of morphin more pronounced than if the patient were free from organic disease.

A number of other cases have been observed by physicians in their daily practice to illustrate the more ready susceptibility to morphin narcosis, of patients who are the subjects of advanced renal disease.[71] This is especially the case in the contracted granular form of nephritis, the amyloid degeneration, atrophy and parenchymatous degeneration of the kidney, the explanation of which is simple. The kidney being the main organ of the body concerned in the elimination of morphin, any obstruction to its eliminating function would obviously allow this poison to remain longer in the system, and, consequently, its poisonous action would be continuously exerted. On this account, the administration of this drug in renal diseases in ordinary medicinal doses may be attended with danger to life which, in normal conditions of health, might not be expected. This is also true in the latter stages of diabetes, when the eliminating power of the kidney is obstructed by a glycogenic degeneration of its lining (epithelial) membrane.

The fact must not be overlooked, too, that the blood may be so surcharged with one poison that a second may not be absorbed until the first has passed out of the circulation; in this view of the matter the symptoms of two drugs may be masked; for instance, the writer has etherized a dog and administered subcutaneously a dose of prussic acid double that required to destroy its life; during three quarters of an hour, while the ether was being inhaled, there was no evidence of poisoning by prussic acid, but, on allowing the dog to recover from the effects of the ether, the poisonous action of prussic

[71] See The Hypodermic Injection of Dangers, by H. H. Kane, M. D., N. Y.; Morphia, Its History, Advantages, and C. L. Bermengham & Co. 1880.

acid became manifested, and the animal died with the usual effects of prussic acid intoxication. Though these latter effects of combination have not been extended to other drugs, yet it cannot be denied that the doubtful question of absorption of two remedies simultaneously in one individual will inevitably form an important factor in the matter of toxicological science. Prof. J. N. Langley,[72] after a very careful series of experiments on the antagonism of certain alkaloid drugs, puts the nature of the antagonism in the following words: "Alkaloids act on the tissues by forming chemical compounds with them. When two poisons act on the same tissue, the result depends on their relative physiological or chemical affinity for the tissue, and the mass of each present. Thus, then, within certain limits to be spoken of presently, if the one alkaloid is given, its effect can be antagonized by giving a sufficient quantity of the other. A limit is placed to this antagonism by the impossibility of giving very large quantities of any substance, without injuring the tissue by physical processes consequent on alteration in the density of the fluids. The antagonistic action of alkaloids can only occur within the limits of doses which do not seriously alter the tissue by altering the normal rate of diffusion, etc. If, for instance, a nearly maximal dose of atropin be given, no other alkaloid we are acquainted with can antagonize its action on the salivary glands, for the chemical affinity of atropin for the tissue is so great that it would require, to compensate for this, a mass of the other alkaloid impossible to be given without direct injury to the tissue. . . . The only case in which a return to normal seems to be possible, is when the poisons combine together, or when they act on some third tissue, as blood, which influences the gland or other structure in question." Now what is true in regard to physiology is true also in regard to toxicology, and, in a given case of poisoning by more than one drug, the expert should always bear in mind that the nature of symptoms must necessarily be influenced by the rules which govern the action of either or both. For a further discussion of this matter the reader is referred to an article by Rossbach;[73] also to the treatise of Fothergill.[74]

Writers on toxicology have usually included in their text a descrip-

[72] Journal of Physiology, August, 1880.

[73] Appendix, Case No. XVIII. Pflüger's Archiv. Bd. XXI. Hf. 1, p. 1, 1879. Rossbach's experiments are not so conclusive as those reported by Langley, and do not affect the question as above stated.

[74] The Antagonism of Therapeutic Agents, by J. Milner Fothergill, M. D.; Edin. Phila., Henry C. Lea, 1878.

tion of the various antidotes which are to be used in cases of poisoning. While there is much to be said in favor of this custom, it should also be mentioned that an ignorant use or, rather, abuse of the antidote may increase the danger to the life of the sufferer rather than act as a cure.

It is of the utmost importance, before resorting to the use of an antidote, that we should be assured what poison has been swallowed. We should not assume, because the effect of the poisonous substance appears to have produced a corrosive action, that an acid corrosive has been ingested; for a caustic alkali may simulate to the inexperienced observer the same local effects upon the mouth and throat. It is quite evident that, if a caustic alkali be inadvertently administered in such a case of corrosion, from the ingestion of an alkali, this treatment would be only an aggravation. The use of a stomach pump, after the caustic alkali has softened the tissues by its prolonged contact with the mucous membrane of the throat and gullet, is certainly not to be recommended; nor would it be advisable to administer an irritant emetic, like mustard, to provoke vomiting. Moreover, it should be remembered that, when using the so-called physiological antidotes, we are handling remedies whose action is used only to counteract an unnatural physiological condition, and to proceed with the latter with the knowledge that we are fighting one, with another, dangerous poison.

47. Suicide; its relation to life insurance.— This relation has become so notorious in recent years from suits at law between life insurance companies and the beneficiaries under the terms of the policies issued to persons whose death has occurred either by suicide or from suspicious circumstances, that it may well suggest more than a passing comment in works on medical jurisprudence. Cases of suspicious deaths have occurred in which others than the victim of suicide have been suspected of conniving at his death. We have mentioned on a previous page (§ 7, *supra*) a case of suicide which the victim attempted to conceal under the guise of death from natural causes. There are well-known cases where a person has committed a self-murder in order to secure a premium effected upon the risk of his life for the benefit of his family. Provided he wished to effect this purpose fraudulently, and to conceal his act of suicide, the victim would seek a means of self-destruction which would escape detection, and to this end he would select a poison whose effects would resemble a natural disease. On this account, therefore, the question of determining a natural from an unnatural death assumes an impor-

tant position, and therefore the physician who is called upon to state the cause of a sudden death should ascertain whether the deceased had taken a policy for life insurance.

We would refer our readers to the case of Mrs. Manson's trial, previously mentioned, for the murder of Mrs. Julia A. Wilson at Putnam, Connecticut, during March, 1903, in which the defense put up the plea of suicide to cover the murder. Another interesting case of supposed suicide of a resident of Newtonville, Massachusetts, concerning which a suit was brought by certain life insurance companies who had issued large insurance upon the life of the decedent, a synopsis of which is presented in our Appendix (case No. XXIX.). In this case the medical examiner, after a post-mortem examination, had given a certificate of the cause of death as due to nephritis complicated with a cardiac affection and congestion of the lungs. Unfortunately for the absolute attestation of a natural disease of the kidneys as explaining the cause of death, though in the first examination the medical examiner's opinion was verified by Dr. Mallory's microscopical investigation of one of the kidneys of the decedent, yet, at a subsequent autopsy on exhumation of the cadaver, the kidney could not be found. If the prominence of the question of fraudulent concealment from the insurance companies had been suspected, more care would have been taken in preserving the proofs of the cause of death. There was no imputation upon the integrity or ability of the medical examiner in this case; but the confirmation of his opinion would have been made clear had the kidney been preserved for a future re-examination. The suspicion aroused was intensified by the fact that much of the large amount of life insurance had recently been obtained, and that, after death, the insured was found without substantial means, and even suspected of having before his death forged documents with which to obtain money to pay his premiums. In the above case most of the life insurance companies paid the full amount, but some compromised by paying 50 per cent of amount insured, which would seem to indicate that there were some grounds to support the theory of suicide. This, and other similar cases, will furnish a lesson to all, that each case in which the official view of a supposed death by suicide is not clearly established by incontestable proofs as caused by the hands of the deceased should be subjected to a judicial inquiry. It is hardly necessary to add that the death in this case was suspected by the agents of the insurance companies to be attributable to some poison taken by the suicide during the night before his death, but could not

be proved on chemical analysis because the contents of the stomach were also lost prior to the exhumation. The attention of the reader is also called to the notes, cited in Appendix, of another case, of the murder of Marcus M. Rogers by his wife for the purpose of obtaining a small amount of life insurance on the supposition that the man committed suicide. Case No. XXVIII., Appendix, p. 790.

48. Classification of poisons.— We propose in the following chapters to present the various poisons under: First, the occurrence in nature, in preparations, in arts and manufacture; second, the properties of the poison; third, symptoms of poisoning,—acute, subacute, chronic; fourth, lethal dose,—action and duration; fifth, diagnosis of poisoning; sixth, treatment by antidotes; seventh, post-mortem appearances; eighth, chemical examination and tests.

Poisons have been variously classified by different authors, but none of the systems is entirely free from objection, either theoretical or practical. Thus they have been classified according to the kingdom from which they are derived, into mineral, animal, and vegetable; also, according to their chemical properties, into organic and inorganic, acids, alkalies, and salts; but perhaps the most satisfactory methods of classification were those based upon their physiological action, the principal ones of which are those of Taylor[75] and Tardieu.[76]

We shall not in this treatise confine ourselves exclusively to any of the systems laid down in books upon poisons, nor shall we, as in our former editions, follow the old classification of Orfila, *viz.*, into irritant, narcotic, and narcotico-irritant, which has become so familiar to American medical jurists; it is by no means free from objections, nor is it strictly accurate. This division cannot be rigorously maintained except for the purpose of convenient reference; since there are poisons usually classed under irritants, which are likewise sometimes narcotic in their action, as, for example, arsenic and oxalic acid; and, on the other hand, some of the pure narcotics, especially opium, occasionally produce the symptoms of irritant poisoning. The classification has been, however, sanctioned by the use of the most eminent toxicologists, but is rather obsolete as shown by more recent knowledge both in chemical and physiological researches.

The classification as given by Taylor is in accordance with the following scheme:

[75] Taylor on Poisons, 1875, p. 73.
[76] Etude Medico-Légale et Clinique sur l'Empoisonnement, Paris, 1875, p. 170.

- **Irritant**
  - Mineral
    - Acid poisons.
    - Alkaline poisons.
    - Nonmetallic.
    - Metallic.
  - Vegetable.
  - Animal.
- **Neurotics**
  - Cerebral.
  - Spinal.
  - Cerebro-spinal.
  - Cerebro-cardiac.

The following is the classification of M. Tardieu: (1) Irritants and corrosives; (2) hypostheniants; (3) stupefiants; (4) narcotics; (5) tetanics.

1. Poisoning by irritant or corrosive poisons has the peculiarity attributed to a local irritant action, which may result in a violent inflammation, corrosion, and a disorganization of those tissues attacked by the poisonous substance; the effects of the substance which has been swallowed being almost exclusively due to lesions of the digestive organs.

2. The hypostheniants have the peculiarity, not merely of producing a local irritation, though this may actually occur, but also of causing general disturbances resulting from their absorption; these latter may be disproportionate to the local effects, which oftentimes are entirely absent, and are very different from irritation or inflammation; they consist in fact of a rapid and profound depression of the vital forces, and are associated with a manifest alteration of the blood.

3. Poisoning by stupefying poisons, of which the largest portion have been comprised under the improper denomination of narcotico-acrid, though producing neither narcotism nor acridity, has, as its essential character, a direct special action upon the nervous system, a depressant action which corresponds to what is called in semiotics (symptoms of disease) stupor, sometimes accompanied with a local irritation, oftentimes of a slight character.

4. Poisoning by narcotics is characterized by an action quite special and distinct, that can only be defined by its own name "narcotism."

5. Finally, poisoning by the tetanic poisons is characterized by a violent excitation of the nervous centers, the intensity of which may be so great as to produce instant death.

The symptoms produced by each class of poisons are sufficiently indicated by their name. Thus, the irritants produce vomiting and

purging, intense abdominal pain, and fatal exhaustion. Septic irritants produce, in addition, symptoms which are known as typhoid; and certain metallic irritants in small and long-continued doses give rise to impaired digestion and nutrition, and death by a gradual exhaustion of the system. Their primary action is expended upon the intestinal tube, causing inflammation or corrosion, and the effect upon the nervous system is secondary. The narcotics produce fulness of the head, vertigo, dimness of vision, delirium, coma, paralysis, and sometimes tetanic convulsions. The narcotico-acrid poisons produce stupor or delirium and convulsions, and also irritate the stomach and bowels, causing vomiting and purging. Both these and the irritant poisons are commonly known by their taste, which is hot and acrid, metallic, nauseous, or bitter; arsenic is the chief exception to this rule, as its taste may be either unperceived, or only distinguished as rough or sweetish. The corrosive poisons, such as the mineral acids and caustics, have so violent an effect upon the mouth and throat that the mere fact of their having been taken affords a good presumption, in this country at least (from the readiness with which any kind of poison can be obtained), against their having been voluntarily swallowed. The pure narcotics have only a slightly disagreeable taste. This class is, however, but a small one compared with the others, and, with the exception of opium, rarely gives rise to judicial investigations.

The large number of poisons, which, since the time of the earlier toxicologists, have been added to poisonous substances, and many of which are in common use, may add to a necessary confusion in any method of the older classifications; yet, attempts at a new method may add still further and more serious confusion. The preceding classifications are so well known that they are almost classic. There are, however, a great many new poisons which it would be difficult to place, in the light of recent scientific knowledge of their physiological action, in either of the above classifications.

We will therefore, in the following pages on the details of the action of poisons, adopt a new system of classification based on the methods which the chemists would use in searching for the poison which would cause the death of the victim subjected to inquiry as to the cause of death. We are fully aware that toxicology has not advanced to such a period of scientific knowledge that any scheme of classification can be in accordance with the line of knowledge of today, and, after a series of years, retain its same position in the group in which we propose at present to arrange it. It would seem.

however, that the groups, as we have arranged them, will make the details of information contained in the following pages of more practical value to the expert who is called upon to present his information as to what poison may have constituted the cause of death in a given case, than if we continued the former classification used in earlier editions.

The most desirable scheme would be a classification based upon the actions of the various poisons; but the science of therapeutics is not sufficiently advanced to render this possible with the desired certainty. Further, many poisons act differently according as to whether they are given in small or large doses, and an empirical classification along these lines would necessitate placing the same poison in more than one class. A classification based according to the origin of the poison has its disadvantages; but they are not as great as those suggested in attempting to classify poisons by their action. A modification of this latter scheme, based more upon the chemical and physical properties of the poisons, together with their nature and origin to a certain extent, seems to us at present the most desirable; *viz.*:—

I. Gaseous poisons (organic and inorganic).
II. Inorganic poisons (solid and liquid).
III. Organic poisons (nonalkaloidal).
IV. Organic poisons (alkaloidal).
V. Food poisoning, ptomains, toxins, leukomains.

This classification appeals to the chemist, as it is along these lines that he conducts his analysis, and it is often called, therefore, the analytical classification.

CHAPTER II.

GASEOUS POISONS.

I. Carbon dioxid.
49. Occurrence of carbon dioxid.
50. Properties.
51. Symptoms.
52. Treatment.
53. Post-mortem appearances.
54. Tests for presence of carbon dioxid.
II. Carbon monoxid.
55. Occurrence, in general.
56. Occurrence in illuminating gas.
57. Other occurrences.
58. Properties.
59. Symptoms of acute poisoning.
60. Action and duration.
61. Lethal dose.
62. Treatment.
63. Post-mortem appearances.
64. Examination of blood.
65. Tests.
III. Sulphid of hydrogen, hydric sulphid, sulphureted hydrogen, sewer gas, hydrogen disulphid.
66. Occurrence.
67. Properties.
68. Action.
69. Symptoms.
70. Chronic poisoning.
71. Lethal dose.
72. Post-mortem appearances.
73. Tests.
IV. Nitrous oxid, protoxid of nitrogen, nitrogen monoxid.
74. In general.

I. Carbon dioxid.

49. Occurrence of carbon dioxid.—Carbon dioxid is one of the most common gases, as it is of universal occurrence. Respiration of all animals, processes of decay and fermentation, and processes of burning, are all accompanied by the production of this gas.

50. Properties.—Carbon dioxid, or carbonic acid gas, when heated, is heavier than common air (sp. gr. 1.52 as compared with

air), and will therefore be found in greatest quantity near the floor after combustion has ceased; but during combustion, or while the air is still warm, it will be equally diffused through the apartment.

Carbon dioxid, formerly called carbonic acid, is not strictly a poison, though the inhalation of pure carbon dioxid gas causes death by preventing the elimination of the same gas, and also by depriving the system of oxygen, thus causing death by asphyxia.

M. Demarquay[1] states from his own observation and experiments, if carbonic acid is injected into the veins, taking care not to inject with such force as, by entering the cavities of the heart to cause death by mechanical distention of that organ, it is absorbed in large quantity and quickly eliminated. Introduced into the system by the lungs, carbon dioxid produces none of the toxic properties so commonly attributed to it. In fact the mammalia can breathe a long time, without any serious inconvenience, atmospheric air or oxygen one fourth or one fifth of whose volume consists of carbonic acid. In man a slight disturbance may be caused after the lapse of a time, varying with the susceptibility of individuals, but lasting long enough to cause the development of certain therapeutical effects which have indicated the use of the agent. Finally, the lesions after death caused by the inhalation of this gas, whether in man or animals, do not in the least resemble those caused by the toxic agent carbon monoxid, formerly called carbonic oxid, with which it has often been confounded.

Most of the accidents caused by coal gas, confined air, vapors from vats, laying aside all account of carbonic acid, should be in a large measure attributed either to carbonic oxid, sulphid of hydrogen, alcoholic fumes, or even to other gases, as yet little known, which have their origin from these sources.

Carbon dioxid, carbonic acid, is simply irrespirable. It is, however, unlike nitrogen or hydrogen in this respect, and is more injurious than these gases because it prevents the inspiration of oxygen and the necessary respiratory functions. Respiration essentially consists in the interchange of gases between the blood and air, and, from the fact that this interchange cannot take place, more in consequence of an absence of the necessary amount of oxygen in an air saturated with carbonic acid, than from the poisonous character of this gas, according to the physical law of gases, it may be seen that the inspira-

[1] Essai de Pneumatologie Médicale; Recherches, etc., sur les Gaz. Paris, 1866, p. 458.

tion of pure carbonic acid is a material obstacle to the respiratory process, and consequently produces asphyxia. On the other hand, hydrogen and nitrogen cannot supply the deficiency of oxygen, and cause the normal changes in the blood necessary to support life, but this is not the result from the same physical law of diffusion of gases, because they are of a different nature from the eliminated gases; yet this interchange can go on only for a limited period, and thus disturbances may be caused that are inconsistent with life.

Carbon dioxid exists from emanations of the soil in rock caves, and is also a product of fermentation of malt and other chemical processes; the well-known precaution of testing for its presence in deep holes, by the lowering of a lighted candle to learn if the gas is present in sufficient quantity to extinguish the light, is familiar to everyone. A candle will burn in an atmosphere of which 10 to 12 per cent of the volume consists of carbon dioxid. In ordinary pure air the proportion is about 4 per cent per volume; when it exceeds this proportion it is irrespirable, not because this quantity of the gas itself is injurious, but because in an apartment which is closely packed with beings who are exhaling from their lungs and the surfaces of their bodies volatile, effete materials are found which are deleterious when inspired into the lungs, and so pass into the human tissues. Therefore, the estimation of the proportional amount of carbon dioxid in a room is often used to determine the extent of the foulness of the air of that room.

When pure carbon dioxid is inhaled, the glottis closes and prevents further inspiration, and the head feels crowded with the sensation of a ringing in the ears, vertigo, relaxation of the muscles, sleepiness, and loss of consciousness. If the inspiration be continued, signs of asphyxia appear, from which the patient may die if he be not allowed to breathe air. According to some experiments of Demarquay, if the body of a person be placed in a tight vat of pure carbonic acid gas, and he be allowed to breathe fresh air, and provided no air is allowed to touch his skin below the neck, he will experience the same sensations as if inspiring the gas through the lungs. Dr. Luff quotes in his book, to which we have previously referred, two fatal cases: "Taylor and Stevenson mention the case of a boy who fell into a vat among some wet hops, and speedily died from respiring the atmosphere of carbon dioxid; two men attempted to rescue the boy, but died in the attempt. A man descended into a large vat, having previously applied the candle test; he was heard to cry out, 'There is gas here,' and instantly fell back dead." He also

relates a case reported by Biggam,[2] where carbon dioxid from a near-by mine emanated from the ground under a house, during a prolonged period of frosty weather, and passed into it. On arriving at the house he found one girl of nineteen dead, and the occupants of the house suffering from the following symptoms: Intense headache, giddiness, nausea, vomiting, and pain in the stomach.

The local action of carbon dioxid on the skin, as shown by the experiments of Demarquay, are anesthetic; he states that in olden times anesthesia was brought about by a friction on the surface of the body of marble dust and vinegar.[2a] It has long been known that ciliary movements are stopped in the presence of this gas. Prof. Jeffries Wyman, of Harvard University, was in the habit of demonstrating this to his pupils in class. Inspiration of this gas will change the red blood in the arteries to black or venous blood. Muscle fiber loses its power to contract when immersed in carbon dioxid. This gas may be absorbed into the economy by exposure to any mucous surfaces or by the skin, when protected from the atmospheric air, or by its introduction under the skin. As it is an acid, it may, by its presence in the blood, convert the carbonates into the acid carbonates (bicarbonates). Its elimination is accomplished quite actively by means of the lungs expiring it into a pure air, by being discharged with the urine, perspiration, and the intestines; consequently when absorbed by drinking water aerated with this acid, its rapid elimination by the digestive tract is easily effected.

Death, after continuous respiration of carbon dioxid, is most probably caused by the presence of this gas, to the exclusion of free oxygen, in the whole of the tissues, thus preventing the interchanges of these gases, called "metabolism," within these tissues, and so causing death by lack of oxidation. This action would destroy all reflex response in the nervous system.[3]

51. Symptoms.—The sudden death by respiration of this irrespirable gas is portrayed so admirably by Prof. Doremus[4] that we quote the picture of symptoms in his own words: "If erect, the person falls to the ground as if struck; the body collapses, the head falling on the breast. The breathing, at first stertorous, becomes suspended. Convulsions may supervene, but not when sufficient oxygen is present. The action of the heart, at first violent, ceases; sensibility is lost and coma follows. The body remains warm, the limbs flaccid, although

[2] Br. Med. Jour. 1893.

[2a] The result of which is evolution of carbon dioxid from chemical action.

[3] Consult Kobert, Lehrbuch der Intoxicationem, Stuttgart, 1893, p. 639.

[4] Peterson and Haines, op. cit. p. 646.

occasionally rigid or convulsed. The countenance assumes a livid color (unless carbon monoxid is likewise present),—especially noticeable about the eyelids, lips, and throat. Sometimes, however, the face is pale and the features are placid. Death occurs without a struggle. Those resuscitated have complained of pain in the head, or soreness of body lasting some days. Paralysis of the facial muscles has ensued. Persons may become gradually accustomed to the gas, and continue to breathe an atmosphere that, to anyone entering, would be unbearable." To support his last statement we would refer our readers to an experiment performed by Prof. Claude Bernard in one of his lectures:[5] He placed a pigeon under a bell glass, and left him to inspire the products of his own exhalations; then he introduced another pigeon to breathe these same exhalations, with the result that the second pigeon died within a few minutes with signs of asphyxia, while the first pigeon continued to live some minutes longer, having breathed during two hours its own exhalations.

52. Treatment.— The person overcome by intoxication of his tissues with carbon dioxid should be placed in an atmosphere of pure air, and laid out upon his back; if his breathing is slow and difficult, artificial respiration should be practised, as in the rules given for a person rescued from drowning; oxygen, now so universally accessible, should be administered for respiration. Irritation of the surface of the body by friction, slapping of the skin with a towel, warmth,—all should be applied; and if the patient can swallow brandy, this, or some other alcoholic stimulant, should be administered. Persistence in these measures of relief should be continued even when, apparently, the patient has ceased to breathe and the pulsations of the heart are almost imperceptible, because recoveries are known to have been made under these circumstances.

53. Post-mortem appearances.—The usual effects of asphyxia, recognized after death by drowning, are observed in death from this gas, namely, engorgement of the venous system, and the right ventricle filled with dark blood in a fluid state, while the vessels of the brain and lungs are congested. The blood in the arteries, if any is found therein, is usually of a dark color, and is not red and rutilant.

54. Tests for presence of carbon dioxid.— The most common test for the presence of considerable quantity of carbon dioxid in a suspected well or confined air space is the dimming or extinguishing of a candle flame when put into such an atmosphere. The least dimming should

[5] Lecons sur les effets des Substances toxiques et medicamenteuses, p. 119; Baillière et Fils, Paris, 1857.

warn a person against entering such an atmosphere. Twelve to fifteen per cent[5a] of carbon dioxid in the air generally extinguishes the flame. When carbon dioxid, or air containing it, is shaken with or drawn through a solution of baryta water or lime water, a turbidity due to the formation of $BaCO_3$ or $CaCO_3$ is produced. This $BaCO_3$ or $CaCO_3$ dissolves in dilute hydrochloric acid with effervescence. A quantitative estimation of the amount of carbon dioxid may be made by shaking a given amount of the air, 5 to 6 liters, in a bottle with 50 c.c. of a standard $BaO_2\ H_2$ solution, filtering off the precipitated $BaCO_3$ in a bell jar in which is a beaker of KOH or NaOH to absorb any CO_2, and then titrating the filtrate against a standard acid solution and determining the loss of alkalinity due to the CO_2.[5b] Large amounts of CO_2 may be determined by drawing air, previously dried by passing it through H_2SO_4, through a carefully weighed vessel containing concentrated KOH solution, when any increase in weight will be the amount of CO_2 in the volume of air used.

II. Carbon monoxid.

55. Occurrence, in general.— The most common occurrence of carbon monoxid is its production in the burning of coal. When coal or coke (carbon) is burned with an insufficient supply of air or oxygen, carbon monoxid is formed in large quantities, in contradistinction to the combustion of carbonaceous material with sufficient oxygen supply; in the latter case the dioxid is formed in larger quantities, the amount of carbon monoxid being much less. Any combustion occurring in the presence of a small supply of oxygen, such as in furnaces with the dampers nearly closed, is accompanied by the production of carbon monoxid. Also the slow burning of wood or charcoal in confined, closed rooms produces much carbon monoxid, and has led to many cases of death, both suicidal and accidental. At the present day large quantities of carbon monoxid are produced in the manufacture of ordinary illuminating gas as it is supplied in the mains of most large cities. Water gas, which is mixed with illuminating gas in large quantities, contains often as much as 30 to 40 per cent carbon monoxid.

In the Massachusetts returns (for 1890) from medical examiners, one case is reported of fatal poisoning by carbon monoxid from the inhalation of charcoal fumes, also two fatal cases (1893) of persons sleeping near lime kilns.

5a Peterson and Haines Txbk. L. M. and Tox. Vol. II. p. 647.

5b Williams. Commemoratory Vol. Univ. College, Sheffield, 1897, p. 132.

Carbon monoxid, formerly known as carbonic oxid, was at one time supposed to pass through the red-hot iron in a cast-iron stove or furnace. Within later years this has been proved[6] by Remsen not to be true, but instead that this coal gas passes out of these coal heaters into the air of an apartment, owing to their faulty construction, or to the pressure from the evolved gas by the combustion at a low heat inside the stove, under the deposit of burnt coal ashes. The experience of taste which this produces in the mouth is well known to one who has been near the stove under these conditions. This is also called furnace gas. This coal gas is also produced from burning charcoal,—especially at a low point of combustion. It is also produced when a low, red heat is given to anthracite coal or coke in a closed generator, as in the so-called manufacture of water gas. When in this condition of combustion steam is forced into the closed generator, and a mixed gas of carbon dioxid and carbon monoxid is made very rapidly under pressure in the generator; this is called by gasmakers "blue gas," because it burns with a pale blue flame. The temperature at which this coal gas is produced is about 1000° C. (1832° F.)

On the continent of Europe, and especially in Paris, self-destruction by the vapors of charcoal is one of the most common forms of suicide. In England and the United States this agent is seldom resorted to, while accidental death from the gases escaping from burning coal or the smothered combustion of wood is very frequent. Dr. Taylor found by experiment that, in burning a quantity of charcoal actively in an open brazier raised above the floor in a large apartment, the proportion of carbon monoxid was nearly equal in air taken a foot above and a foot below the level of the source of combustion, there being no current to affect the results. The inferences which he draws from this and from other considerations are: (1) That, in a small and close apartment, individuals are equally liable to be suffocated at all levels, from the very equal and rapid diffusion of carbon monoxid during combustion. (2) That, in a large apartment, unless the gas be very rapidly diffused by a current of air, the air around the source of combustion may become impregnated with a poisonous proportion, while that at a distance may still be capable of supporting life, because carbon monoxid requires time for its perfect and equable diffusion in a very large space.[7]

[6] National Board of Health Bulletin, Washington, 1881, Vol. II. No. 52, p. 857.

[7] Med. Jur. p. 535.

The following case may serve to show the circuitous route by which carbon monoxid may find its way into bedrooms:[8] A man and his wife were found dead in their bedroom; the first in an easy, bent position on his right side, on the floor; the latter in a similar position, and her countenance wore a mild and placid expression. No marks of violence were found upon the bodies, and with the exception of slight suggillations on the man's back, the skin was perfectly natural in color and appearance. A post-mortem examination and a chemical analysis were made without any indication of poisoning being detected. There was a singular and intolerable smell in the house, strongest in the chamber. It was found, upon further inquiry and examination, that a straw mattress had been burned in the cesspool of an adjoining yard a few days previously, the embers of which were still in a state of ignition, and, when stirred, gave off dense volumes of smoke, and a disgusting smell resembling that in the house. The walls of this cesspool and of the foundation of the house were of loose stones, and, under the influence of a strong west wind, the products of combustion had found their way through the foundation into the boarded walls of the house, and thence into the chamber. No sulphureted hydrogen could be detected in the gas which still escaped into the room, but sufficient carbon monoxid to fatally contaminate its atmosphere. Briand enumerates several instances in which carbon monoxid, coming from fires lit in an apartment other than that occupied by the deceased, has, nevertheless, penetrated into it, and been the cause of fatal accidents.[9] In one of these cases a man and his wife were found dead in bed, suffocated by gas produced by the charred woodwork in the neighborhood of a fire in a room at the opposite end of a long corridor on the same floor. The gas had worked its way under the floor until it found a vent in a crack of the flooring in their appartment. In other instances the gas was driven through stovepipes, and from one chimney flue to another on different floors.

56. Occurrence in illuminating gas.— The ordinary illuminating gas, which consists chiefly of light carbureted hydrogen, contains also vapors of volatile, liquid carburets of hydrogen, carbon monoxid (CO), carbon dioxid (CO_2), and other elements. Light carbureted hydrogen is in itself hardly poisonous; but the composite gas, which is now everywhere so freely used for burning, has frequently caused fatal accidents. Still, the atmosphere may be very offensively loaded with

[8] An account of two cases of poisoning with carbon monoxid under remarkable circumstances, communicated by Jos. Law, Esq. Surgeon, etc. Ed. Month. Jour. March, 1853.

[9] Med. Leg. p. 418.

it, and yet be breathed for a short time with impunity. It does not appear to act merely as an asphyxiating agent, but rather like a narcotic.

The danger arising from the inhalation of illuminating gas varies with the kind of gas and with the thoroughness of its purification; the principal danger arises from the carbon monoxid (carbonic oxid) which the gas contains. Ordinary illuminating gas, made from bituminous coal and well purified, consists chiefly of hydrogen (40–50 per cent), marsh gas (35–40 per cent), carbon monoxid (4½–7½ per cent), olefiant gas and other hydrocarbons (4–8 per cent), and usually very small amounts of carbonic acid gas and air. The so-called water gas, made by passing steam over anthracite coal heated to a white heat, yields a gas which burns with an almost colorless flame and contains carbon monoxid (CO). This is mixed with a highly luminous gas, made from naptha, petroleum, or cannel coal, to give it illuminating properties; but even then it contains a very large percentage of carbon monoxid, which is one of the most active of poisons, producing, when inhaled, speedy death, and, according to Leblanc, "one volume of it diffused through 100 volumes of air totally unfits it to sustain life; and it appears that the lamentable accidents, which too frequently occur from burning charcoal or coke in braziers and chafing dishes in close rooms, result from the poisonous effects of the small quantity of carbon monoxid (CO) which is produced and escapes combustion, since the amount of carbon dioxid thus diffused through the air is not sufficient in many cases to account for the fatal result."[10] When it was proposed to supply Les Invalides in Paris with water gas, a commission was appointed, consisting of Messrs. Dumas, Chevreul, and Regnault, eminent chemists, to investigate it. They found that it contained from 30 to 40 per cent of carbon monoxid (CO), and reported "that it would be dangerous to the occupants of the institution to introduce, even by way of experiment, gas obtained from the decomposition of water by the Kirkham process."[11] This gas was the odorless carbon monoxid and hydrogen mixture. H. Letheby says:[12] "Seligue, in 1840, obtained permission to use the gas in the towns of Dijon, Strasburg, Antwerp, and two of the faubourgs of Paris, and Lyons. At Strasburg an accident occurred which put a stop to its use. The gas escaped from the pipes into a baker's shop,

[10] Bloxam's Chemistry, p. 118.
[11] London Journal of Gas Lighting, May 20, 1862.
[12] London Journal of Gas Lighting. June 10, 1856.

and was fatal to several persons; and not long after, an aeronaut, named Delcourt, incautiously used the gas for inflating his balloon. He became insensible in the car, and those who approached to render him assistance fainted and fell likewise. The use of the gas has therefore been interdicted on the Continent."

Since the above was written in the previous edition of this book, there has ensued an increase in the use of enriched "water gas," not only in the cities of the United States, but in the large cities of Europe; thus, more than a passing notice should be given to the noxious agent contained in this gas.

The danger of inhaling this gas is great, when, through leaks, it may permeate sleeping chambers and apartments of houses; this has therefore caused a sentiment of alarm,—especially in Massachusetts, where a watchful gas commission has for years been charged with legislative functions to investigate all deaths from accident resulting from inhalation of gas.[13] Previous to 1888 the laws of that state had forbidden the distribution of an illuminating gas which contained more than 7 per cent of carbonic oxid.* The report of that commission is as follows:—

1888	3	cases	with	recovery.
1889	2	fatal	2	recoveries.
1890	8	"	9	"
1891	11	"	11	"
1892	18	"	3	"
1893	30	"	22	"
1894	32	"	37	"
1895	35	"	41	"
1896	51	"	44	"
1897	60	"	74	"
1898	77	"	45	"
1899	65	"	69	"
1900	47	"	52	"
1901	36	"	64	"
1902†	61	"	138	"

*About three quarters of gas distributed in Boston changed to coal gas.

†During this year much less coal gas was distributed in Boston on account of scarcity of coal.

About the beginning of 1900 a very large proportion of the

[13] Extract from an article by the writer published in Vol. III. Public Health Papers of the American Public Health Association.

gas distributed in the city of Boston was changed from water gas to a gas made from distillation of bituminous coal, which contained a much smaller proportion of carbon monoxid (CO).

The published reports of this gas commission contain some interesting *data,* which it gathered from inquiries made in other parts of the United States, as follows:—

Thirty-five deaths from suicide and 139 by accident in 1890 (included in these, 1 by oil gas, 6 by coal gas, 49 by water gas, 83 by the last two mixed gases); 33 by suicide and 178 by accident in 1891; 37 by suicide and 213 by accident in 1892.

These reports contain, also, the following *data* gleaned from the cases reported from their own state:—

Two years (1900–1), fatal cases from water gas, 22, mixed coal and water gas, 51, coal gas, 4; same years, nonfatal cases from water gas, 22, mixed coal and water gas, 51, coal gas, 8; in 1902, fatal cases from water gas, 16, mixed coal and water gas, 61, coal gas, 6; in 1902, nonfatal cases from water gas, 5, mixed coal and water gas, 122, coal gas, 9, oil gas, 1.

The presence of carbon monoxid in illuminating gas, as distributed in many of the large cities of the United States, made from anthracite coal or refuse carbon, from the distillation of petroleum oil and from coals, and enriched with the petroleum oil products, is shown by the preceding analysis of the number of accidents arising from its inhalation, reported by the Massachusetts gas commission, to have caused the death of about half the victims exposed.

The composition of the two usual kinds of illuminating gas, coal and enriched "water gas," taken by the personal experience of the writer in actual manufacture of these two forms of illuminating gas, is as follows:—

Bituminous coal gas,	sp. gr. .490 to .510,	18.3 candle power.
Enriched water gas,	sp. gr. .670 to .673,	26.0 candle power.
Bituminous coal gas contains	2 to 4	per cent CO_2.
Bituminous coal gas "	7 to 7.3	per cent CO.
Nonenriched water gas "	25.3	per cent CO.
Enriched water gas "	25.1 to 27.43	per cent CO, 26.0 c'le p'r.

57. **Other occurrences.**— Prof. Doremus calls attention to another dangerous source from which this very poisonous gas may arise; he has found carbon monoxid escaping from fires made with "smokeless fuel" in briquets; these briquets are used in foot-warmers in street

cabs, in consequence of which the air inside may be heavily charged with carbon monoxid. The symptoms of poisoning by this noxious air were sometimes serious to the occupant.[14]

We have previously recited a case, where (p. 113, *supra*), two persons were found dead in their bed from the inhalation of the products of a slow combustion of a straw mattress in a cesspool, which escaped by means of a loose retaining stone wall into the cellar of the house. When slow-burning flame impinges on soot, this gas is freely evolved; it also arises in fires occurring between flooring, where the woodwork may be charred, and may thus account for the death of a person sleeping in a room on the floor above the combustion,—especially if the ceiling below it is ceiled off by plaster. It is also the product of explosion of most explosives; it may also be distributed in coal dust in a bed of coal. Medical Examiner Hurd reports (Case 1, Returns to Massachusetts secretary of state, 1898) the death of a man, buried under several tons of coal, as caused by asphyxia from coal dust, perhaps from evolution of carbon monoxid.

Prof. Doremus states that "wood gas made by the Wilkinson process was found to contain 33.75 per cent, while the commercial gas, served to consumer and made by mixing the gases from wood, coal, and naphtha, contained 11.25 per cent." He also states that "carbon monoxid does not support combustion, but forms an explosive mixture with one half its volume of oxygen, or two and one half volumes of air."

"Producer" gas, being made somewhat in the same way and from anthracite coal, has about the same proportion of carbon monoxid.

58. Properties.— Carbon monoxid is a colorless gas, of faint odor when pure, almost insoluble in water. It burns with a blue flame. It is lighter than air, having a specific gravity of 0.967. With metals it forms liquid combinations known as carbonyls, which are easily decomposed by heat into the metal and the gas. With air and oxygen it forms explosive mixtures.

59. Symptoms of acute poisoning.— Warm-blooded animals, forced to breathe carbon monoxid mingled with air, show an increase in pressure of the arterial circulation, at the first, which is soon replaced by a diminution in the arterial pulse strength; the surface of the skin becomes mottled with patches of bright-red color, owing to stagnation, or stasis, of the circulation through the capillary vessels which communicate between the arteries and veins. The pulsations of the heart

[14] Peterson and Haines, op. cit. p. 649.

and the respiratory efforts become slower, and the latter stertorous and difficult; reflex response to stimulation of the muscles and to pain becomes less, and finally ceases. In man these symptoms are similar; but, before consciousness is lost, his sensations are headache, vertigo, fainting, anesthesia, and loss of voluntary muscular motion, followed sometimes, but not so often as after inhalation of carbon dioxid (CO_2), by convulsions. The temperature of the body is lowered, and there is a contraction of the involuntary muscles, as shown by an involuntary discharge of feces, urine, and sometimes of semen. Recovery is rapid if the victim is immediately placed where he can breathe pure air. In gas works, where care is taken to watch a workman who is often exposed to danger from leaks of gas, it is very uncommon that the employees suffer serious injury,—especially if the precaution is exercised to have on hand for such emergency a tank of compressed oxygen and some powders of ammonium carbonate, as well as siphons of aerated waters. The effect of the latter appears to distend the walls of the stomach, and so to mechanically excite the eructation of wind and vomiting, and thereby to incite strong efforts to breathe; the mechanical distention of the stomach reacts on the diaphragm. The use of whisky and spirituous drinks is not, in the opinion of the writer, so beneficial a method of treatment as that above indicated, because the alcoholic stimulant is apt to increase the after effects during recovery; these are prolonged headache, nausea, and muscular weakness. Sometimes the effect of intoxication from inhalation of this gas will drive a man into a state of maniacal excitement, which may be dangerous to all about the premises. A case of this kind is known to the writer: This man had been engaged in calking a gas connection to a station meter, and was "overcome" from inhaling the water gas; when pulled out into the air he rushed out with the calking iron in hand, and threatened the bystanders; being left to himself for a while, he gradually recovered his usual quiet manner, and afterwards had no recollection of his violent action.

When death occurs, asphyxia seems to be caused by loss of reflex action, due to the fact that the tissue in the nerve center at the base of the brain (medulla oblongata) has lost its function of inciting respiratory efforts.

60. Action and duration.— Most of the toxicologists incline to the belief that carbon monoxid intoxication produces a definite combination with the hemoglobin of the blood corpuscles. This theory, at first advanced by Tardieu and other toxicologists in France forty

years or more ago, was supported by observing that a mixture of fresh blood with this gas reduced the oxyhemoglobin to carbon-monoxid-hemoglobin, and which condition, by its stable union, will prevent the re-formation of oxyhemoglobin; the monoxid being more stable than the oxy-combination is more slowly eliminated from the tissues, and, by its presence, prevents the normal interchange of gases within these tissues; in consequence, oxidation and the production of the result, carbonic acid combination, do not take place, which is essential to the maintenance of life. If, therefore, a man continues to breathe an atmosphere, according to Grehant's experiments, containing 0.5 carbon monoxid to 1000 parts of oxygen, for one hour, his blood will be a third poorer in oxygen and contain 152 times more carbon monoxid than in the above mixture; in an atmosphere containing 10 per cent carbon monoxid, the character of the blood changes so quickly during ten to twenty seconds that then it will show 4 per cent of carbon monoxid, and, at the end of seventy-five to ninety seconds, it will show 18.4 per cent; breathing, during half an hour, an atmosphere containing 0.07 to 0.12 carbon monoxid will show that a fourth part of the red corpuscles of the blood are incapable of combining with oxygen. The animals subjected to these experiments by M. Grehant never showed a saturation of the blood with carbon monoxid to the exclusion of oxygen; for long before this could take place they were dead.[14a]

Kobert[15] states: "That carbon monoxid has a direct action on the nervous system is shown in a marked manner, when an atmosphere of oxygen with at least 20 per cent carbon monoxid is breathed; for, in the first minute, there is acute cramp or total paralysis of the limbs, when the blood in no way attains the saturation sufficiently great to account for such symptoms. Geppert has, through a special research, shown that an animal suffocated by withdrawal of oxygen increases the number and depth of the respirations; but when the animal is submitted to carbon monoxid, in which case there is quite as much a withdrawal of oxygen as in the former case, yet the animal is not in a condition to strengthen its respiratory movements. Geppert, hence, rightly concludes that carbon monoxid must have a primary specific action on the nerve centers. I (Kobert) am inclined to go a step further, and, on the ground of unpublished researches, to maintain that carbon monoxid not only affects injurious-

[14a]Blyth, op. cit. p. 66.
[15]Lehrbuch der Intoxicationen, p. 526, quoted by Blyth, also, translation by Frieburg.

ly the ganglion cells of the brain, but also the peripheral nerves (for example, the phrenic), as well as divers other tissues, muscles, and glands; and that it causes, so rapidly, such a high degree of degeneration as not to be explained through simple, slow suffocation; even gangrene may be caused."

Gangrene is mentioned by many writers as a sequela of carbon monoxid intoxication, and both sugar and albumin also are found in the urine.

The above explanation of the action of carbon monoxid on the human economy demonstrates, more clearly than the results of Prof. Grehant, those few cases where the life of the victim of intoxication is sometimes prolonged, and ends fatally within a few days with symptoms of disturbance in the functions of the central and peripheral nerve system, such as those described by Prof. Doremus: "Alteration in the digestion, diminished vigor, gray color of the skin, coated tongue, loss of memory, diminution of the psychic powers, and occasional convulsions." This opinion is borne out by the similarity of these symptoms to those which occur in the final stages of diabetes; for it has been observed that, equally with this latter disease, boils and carbuncles are among the sequelæ of carbon monoxid poisoning. This opinion is still further supported by Gruber,[16] and by Sedgewick and Nichols,[17] whose researches would seem to prove that there are not only no accumulated effects of this poisonous gas, but that, when it is inhaled in very small quantities, it disappears from the blood. The presence of this gas may be found in the blood of a person poisoned by its inhalation, if acute, and provided the examination be made almost immediately after death; but in subacute and chronic cases it cannot be found. This is not surprising if the theory of Kobert be correct, namely, that the stoppage of life by this intoxicant is not from the condition of the blood corpuscles, but from the prevention of the activity of the functions of the nerve system, due to the deficient supply of oxygen in its tissue. In other words, during life, and even if there is only a feeble respiration, the combination of carbon-monoxid-hemoglobin is not so stable that it cannot be removed by receiving a gradually increasing amount of free oxygenated air into the lungs.

61. Lethal dose.— According to the above-related experiments of Grehant, and the experiments of Nichols in Massachusetts, air con-

[16] Ueber den Nachweis und die Giftigkeit des Kohlenoxydes, und sein Vorkommen in Wohnräumen, Arch. f. Hygiene, 1883, Vol. I. p. 145.

[17] Sixth Annual Rep. State Bd. of Health, etc. Boston, Mass. 1885.

taining 0.07 to 0.12 of carbon monoxid is fatal in an hour to an animal exposed to inhale it; but there are no definite and accurate calculations of the proportion of carbon monoxid in air, when fatal inhalation of this gas mixed with air has occurred to human beings; generally, the volume of gas is so much higher than a lethal dose that this question has not been raised. It is not at all improbable that many of the nonfatal cases, reported as recoveries, have breathed an atmosphere where the inflow of fresh air has been sufficient to reduce the percentage to one far below those used in the experiments on animals.

62. Treatment.— As before remarked, the relief to a person, overcome with having inhaled air mixed with a dangerous amount of carbon monoxid, should be immediate. The person should be removed to a place where he can breathe fresh and warm air; for, as these accidents occur more frequently in winter than in summer, owing to the freezing of the ground and the permeation of the gas through loose cellar walls into buildings, one naturally, without thinking, will often expose the patient out of doors, when it would be wiser to place him in a warmed room, free from gas, and where a little air may be let in through an opened window. One of the most important objects for relief should be to keep the surface of the body warm, in order to promote active circulation through the blood vessels of the skin and extremities. Respiration of pure, warm air should be actively encouraged, even to induce artificial respiratory motions, rhythmically at fourteen to eighteen times per minute. Compressed oxygen should be obtained if possible; the best stimulant to excite the heart is carbonate of ammonia in weak solution. These measures have, in the experience of a number of the writer's years as manager of a large gas company making both kinds of illuminating gas, served to resuscitate many persons overcome with gas, whose appearance, at first sight, seemed almost hopeless of recovery.

63. Post-mortem appearances.— Schmaus and Ewing state as follows: "After poisoning by carbon monoxid, the blood in the cadaver is a peculiar cherry-red and remains fluid, and the post-mortem *livores* (patches, or intervascular spaces) are also of a bright red." The skin of the face, neck, and of the trunk of the cadaver, shows scattered patches with irregular outlines; but these are not gravitation or post-mortem ecchymoses, because they are not usually observed on the back; they generally appear before death and persist afterwards. These red blotches, characteristic of this poison, are probably due to the stasis of blood in the relaxed capillaries of the

skin, from which the bright red coloring of the blood exudes into and stains the subcutaneous tissue surrounding these vessels. The countenance of the face is placid and calm,—especially after a slow action of the poison. Putrefaction is delayed for a long time after death. When death occurs rapidly in an atmosphere strongly charged with carbon-monoxid gas, the post-mortem appearances of asphyxia, namely, congestion and edema of the lung tissue with effusion, are more marked; when, however, death occurs more slowly in an atmosphere slightly charged with the poison, the congestion or injection of the internal organs in the abdominal regions and of the membranes of the brain is strongly marked. The right side of the heart is generally suffused with blood and the left side is comparatively empty.

64. Examination of blood.— The detection of this form of poisoning after death depends chiefly upon the recognition of the carbon monoxid in the blood by the spectroscope, since the coloring matter of the blood unites with it to form a compound which gives characteristic absorption bands when examined by the spectroscope. In cases of poisoning by carbonic oxid, or any gaseous mixture which contains it, the blood has a bright-red, almost rose, color, which persists several days.[18] This color is due to the union of the blood pigment, hemoglobin, with the carbon monoxid, which has replaced the oxygen normally combined with the hemoglobin, thus depriving blood pigment of one of its physiological functions, *viz.*, that of carrying oxygen through the body. If blood which contains this compound of hemoglobin and carbon monoxid be, after suitable dilution with water, examined with the spectroscope, two absorption bands similar to those of the natural blood pigment will be seen, but differing from them by the fact that, when treated with sulphid of ammonium, they do not disappear but, rather, coalesce; moreover, this compound is a very stable one, and is not destroyed by drying, so that it can be detected, according to Eulenberg and Jaederholm, in blood after it has been dried for several weeks, by moistening again with water and examining with the spectroscope.

65. Tests.—In forensic investigations the most reliable test for the presence of carbon monoxid is its action upon the hemoglobin, the coloring matter of the blood. Hemoglobin has a peculiar affinity for carbon monoxid, in that it resists the ordinary reduction means employed in reducing oxyhemoglobin to hemoglobin. Oxyhemoglobin, the compound of hemoglobin with oxygen as it occurs in the arterial

[18] Huenefeld, Blutproben vor Gericht und das Kohlenoxydblut, Leipzig, 1875.

blood, when examined by means of the spectroscope, shows the presence of one or two absorption bands, according to the dilution. If the solution is concentrated, the only light transmitted is in the orange, the rest of the spectrum being completely absorbed. If this solution is diluted, two bands, one near the D line and the other towards E, make their appearance. With further dilution the band near E disappears. With reducing agents, such as ammonium sulphid or Stokes's reagent, the two bands above mentioned coalesce, forming one broad band, which generally has not sharply outlined edges, and lies between D and E. With carbon-monoxid-hemoglobin solutions in proper dilution, two bands are seen almost in the positions occupied by the two bands of oxyhemoglobin, but easily distinguishable from them by their remaining unchanged on the addition of reducing substances to the solution. Carbon-monoxid-hemoglobin is reduced only with difficulty and on long standing, whereas oxyhemoglobin is easily reduced to hemoglobin under the same conditions. In examining minute quantities of blood for the presence of carbon-monoxid-hemoglobin, the use of the microspectroscope may be employed to advantage.

III. Sulphid of hydrogen, hydric sulphid, sulphureted hydrogen, sewer gas, hydrogen disulphid.

66. Occurrence.— Sulphureted hydrogen is formed in the putrefaction of organic matter containing sulphur, and in the body,—especially in the intestines. Vegetable and organic matter, such as in sewers and cesspools, gives rise to considerable quantities. Anaerobic bacteria produce this gas from the decaying vegetable matter at the bottom of ponds or lakes, and the gas rises to the surface, tending to discolor any buildings in the neighborhood painted with white lead.[19]

67. Properties.— A colorless gas (sp. gr. 1.19) of a peculiar, nauseating odor, quite soluble in water, acid reaction, combustible, burning with a blue flame, with the production of sulphur dioxid and water.

68. Action.— This is the principal deleterious gas which is evolved from privy wells, and from foul drains and sewers. Its familiar and extremely offensive odor affords such unmistakable evidence of its presence that, unless a person is obliged to inhale it, or is exposed to it in a concentrated form, accidents will rarely occur from it. The consideration of its effects, and the means of obviating them, are evi-

[19] Peterson and Haines, op. cit. p. 380.

dently more the subjects of medical police or hygiene than of legal medicine. A few observations may not, however, be misplaced. When not existing in a very large proportion in the atmosphere, it may be breathed for a certain time with comparative impunity, giving rise merely to lassitude, loss of appetite, and sometimes a typhoid febrile condition. Again, when inhaled in a greater quantity, the symptoms are an acute and oppressive pain in the head and pit of the stomach; and for this reason, this gas has received from the French the name of *"plomb des fosses."* If, after experiencing these sensations, the individual does not immediately withdraw from his position, he loses his consciousness, and falls, completely deprived of sensibility and the power of motion; a reddish froth runs from the mouth, the body is cold, and the face livid; the eyes are dull, and the pupils dilated and immovable; the pulse very irregular, and almost imperceptible; convulsions ensue, and the person dies comatose.

In a case reported by Dr. Radcliff, of Baltimore, a man who descended into a privy sink nearly eighty feet deep, already almost emptied by machinery, was overcome by the gases and fell to the bottom, where he remained for two hours before he could be extricated. Meanwhile cold water was repeatedly thrown upon him. Fifteen minutes afterwards he was much asphyxiated and depressed, with hurried and difficult respiration, but he recovered rapidly.[20]

Accidents from inhalation of this gas, which is used as a reagent in chemical laboratories, have sometimes been caused to students and others by inhaling its fumes. Cahn reports such an accident to a student, who made H_2S in a laboratory, and inhaled the gas for two hours; he went home to dinner; the symptoms of intoxication did not begin for an hour after he had left the laboratory, and had been breathing fresh air.

69. Symptoms.— The first symptom is a smarting sensation of the eyes, which is followed by difficult breathing, cough, palpitation of heart, chills, muscular weakness, faintness, headache, and cold sweating. These symptoms appear in half an hour after breathing an atmosphere in which the proportion of the hydric sulphid gas amounts to 0.5 per 1000 volumes of air. Next after the local irritation, the symptoms, observed both in man and the lower animals that inhale 0.4 to 0.8 parts in 1000 volumes of air, consist of convulsions and death from signs of pulmonary inflammation, accompanied with a paralysis of the nerve centers. Sometimes the symptoms may simu-

[20] Am. Jour. of Med. Sci. Oct. 1858, p. 377.

late those of typhoid fever; sometimes tetanus and delirium have been observed; these last-named symptoms are caused by a dose of larger amount than above indicated. Sugar has been found in the urine after the attack.

70. Chronic poisoning.— This is more liable to occur to laborers working in sewer trenches, though some become inured by frequent contact with this sewer gas. The symptoms of chronic poisoning from hydric sulphid are conjunctivitis and headache, and often chronic disturbance of digestion. The skin will become attacked by boils; and chemists who work in an atmosphere charged with this gas experience a continued headache with nervous derangements. Several years ago the writer had occasion to pass through the sewers of Boston, before the building of the new system of sewerage was inaugurated. The sewer-gas odor was almost imperceptible because the sewers had been ventilated by the removal of the manhole covers on a bright day early in the month of November when there was a strong westerly wind blowing. He walked through about a mile of the system, and was taken at night with a severe colic; another physician in the party had the same experience; both had chills, and for a few days afterwards suffered from indigestion and nervous derangements.

The above is not unfrequently an experience of those unused to inhaling sewer gas, when subjected to even an exposure of a very diluted air impregnated with sewer gas.

Like many other of the volatile poisons, hydric sulphid has a specific action upon the hemoglobin of the blood corpuscles, and reduces the oxyhemoglobin to a combination called sulphmethemoglobin, which can be recognized by its absorption band in the solar spectrum.

71. Lethal dose.—According to Lehman,[21] air containing in volume 1 to 3 parts to 1000, will kill cats and rabbits within ten minutes; he also states that the utmost that man can breathe of this impregnated air without danger is only a half of this proportion. Letheby makes the statement that one part of hydric sulphid to 100 parts of air would be destructive to human life, and even two per cent may have a poisonous effect.

72. Post-mortem appearances.— When death suddenly takes place within two or three minutes from exposure to this gas, no special post-mortem signs are observed. After chronic poisoning, however, there may be noticed, according to Lehman, edema of the lungs, and

[21] Arch. f. Hygiene. Bd. XIV. 1892, 135.

usually an earlier appearance of the post-mortem green discoloration over the whole surface of the skin, including the face and neck, which is so often observed to follow death from any lingering disease. Unfortunately for the establishment of a positive proof of death by inhalation of this poisonous gas, the spectroscopic test of the presence in the blood of the peculiar band of sulphmethemoglobin is often observed in a cadaver; this alteration is the consequence of a putrescent decomposition of the blood after many natural diseases. Post-mortem signs usually described are the following: A proneness to rapid putrefaction; an offensive odor from all parts of the body; the blood dark and liquid; the right side of the heart congested; the muscles of the body of a dark color, and insusceptible to the stimulus of galvanism.

This is not the only noxious gas evolved from privies and drains, but it is that which is the most destructive to life. There are also ammoniacal emanations, which are extremely irritating to the respiratory mucous membrane; and nitrogen gas, which sometimes accumulates in enormous quantity, but which, although irrespirable, is perhaps not in itself noxious.[22]

73. Tests.— The odor is very characteristic, and may easily be detected one part in 10,000,[22a] and often in greater dilution. A dilute solution of lead acetate will give a black or brown precipitate or coloration when the gas is passed through such a solution, 1:250,000.[22b] Cadmium and arsenic solution acid with acetic acid will give the yellow precipitates of CdS or $As_2 S_3$. Sulphureted hydrogen solutions, when treated with 1/50 of their volume of concentrated HCl and a few milligrams of para-amido-meth-anilin sulphate and one or two drops of a dilute ferric chlorid solution, give a blue color (Caro Fischer test). This reaction is very delicate and is sensitive for .00009–.0000182 grams H_2S per liter.[22c]

IV. Nitrous oxid, protoxid of nitrogen, nitrogen monoxid.

74. In general.— A colorless gas with an odor and taste more or less sweetish, soluble in water, supports combustion (sp. gr. 1.53), and can be easily liquefied.

This gas, more commonly named protoxid of nitrogen, has been

[22] *Vide* Brand, Med. Leg.

[22a] Peterson and Haines, op. cit. Vol. II. p. 671.

[22b] Wormley. Micro-chemistry of Poisons, 2d ed. 1885, p. 372.

[22c] Dragendorff. Ermittelung von Giften, 4th ed. 1895, p. 64.

used very extensively by dentists, and somewhat by surgeons for minor operations as an anesthetic. It deserves but little mention here. Its action is due, most probably, to the property which it possesses of supplying the place of a portion of the oxygen in the blood, with an innocuous respirable gas incapable of supporting life, but not directly interfering with the elimination of carbonic acid gas. If pure, and properly administered, this gas can do no harm unless the inhalation be prolonged beyond the period that respiratory movements are manifested; even then the removal of the inhaler may be immediately followed by inspiration of air, which soon takes its proper place in the lungs and blood, producing the normal changes necessary to the support of life. In this respect it resembles ether, which may also cause asphyxia if administered by an inexperienced person. If, in making the gas, too great heat be used, deutoxid of nitrogen or poisonous nitrous fumes may be formed in the retort holding the nitrate of ammonium (the substance most commonly used in the manufacture of the gas). Yet this is quite soluble in water, through which the gas is generally, and should always be, conducted before passing into the gasometer. The present plan of condensing nitrous oxid and confining the condensed gas in an iron chamber avoids the risk which might occur from the decomposition or fouling of the water, when kept in gasometers.[23] There has been as yet no reliable record of death immediately caused by the use of this anesthetic gas, excepting what may be explained as due to cardiac or other coexisting disease condition. This gas is now often used to produce the so-called pre-anesthetic effect, before submitting a patient to continuous etherization for surgical operations. In Massachusetts returns for medical examiners for 1885, there is one case of suicide reported on the self-administration of this gas.

[23] For further particulars with regard to this gas, reference may be made to Dental Cosmos, Jan. 1869; Med. Gazette, N. Y. (extract from the London Lancet) Dec. 11, 1869; Gazette Hebdomadaire, Paris, Dec. 10, 1869; Dict. Enc. des Sciences Medicales, tome VII. p. 690: Boston Med. and Surg. Jour. N. S. Vol. V. p. 91; Edin. Med. Jour. Jan. 1871; N. Y. Med. Jour. Aug. 1870. This last gives in detail the results of the writer's certain experiments, from which the above text is a summary. Am. Jour. of Med. Sci. 1870, p. 61.

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I. Sulphuric acid.

75. In general.— Sulphuric acid is widely used in the arts and manufactures, and there is hardly a chemical process that does not involve its use in some form or other. It is often used by the poorer classes as a common poison, for suicidal purposes,—especially where difficulties are thrown in the way of procuring alkaloidal poisons.

76. Properties.—Pure concentrated sulphuric acid is an oily, colorless liquid, having a specific gravity of 1.84 or less, according to its strength. (100 per cent acid has a sp. gr. 1.848 at 12° C.) It is odorless, nonfuming, extremely bitter, and is exceedingly hygroscopic, blackening or charring organic substances. The ordinary commercial sulphuric acid generally varies in color from a light brown to black,

due to small amounts of carbonaceous matter and contains traces of lead and arsenic. Great heat is evolved on the addition of water, and the acid should always be added to the water to avoid injury. It dissolves most metals, and if used in a dilute form the sulphate of the metal is formed, and hydrogen gas evolved; if, however, a metal be heated with the strong acid, sulphur dioxid is given off. Fuming sulphuric acid, often called Nordhausen acid, is a colorless (when pure), heavy, oily liquid, formed by distilling $FeSO_4$ and collecting the fumes in concentrated H_2SO_4; it is often considered a solution of SO_3 in H_2SO_4, and has the formula $H_2S_2O_7$. The dilute H_2SO_4 of the U. S. P. is 10 per cent and the aromatic 20 per cent.

Sulphuric acid (U. S. P.), also known as hydric sulphate, oil of vitriol, H_2SO_4, occurs as a "colorless liquid of an oily appearance, strongly caustic and corrosive, and having a strongly acid reaction. Its specific gravity should not be below 1.840. It is miscible in all proportions with water and alcohol, with evolution of heat. When heated on platinum foil, it is vaporized without leaving a residue. If the acid be warmed with sugar, it will blacken the latter; if diluted with 5 volumes of water, the liquid yields, with test solution of barium chlorid, a white precipitate insoluble in hydrochloric acid. . . . To neutralize 2.45 grams of sulphuric acid diluted with about 10 volumes of water should require not less than 48 cubic centimeters of the volumetric solution of soda."

77. Preparations.— (U. S. P.) Acidum sulphuricum aromaticum. Acidum sulphuricum dilutum. Aromatic sulphuric acid contains 200 parts of the pure acid, 45 parts of tincture of ginger, oil of cinnamon 1 part, and a sufficient amount of alcohol to make 1,000 parts. Dilute sulphuric acid contains 1 part of the pure acid to 9 parts of water.

The strong sulphuric acid of the manufacturer contains 100 per cent of acid. The ordinary brown commercial acid, impure, and containing organic matter with lead and arsenic in solution, has a specific gravity of about 1.750, and generally contains 67.95 of anhydrous acid $SO_3 = 85.42$ of hydric sulphate.

Concentrated sulphuric acid absorbs water from the surrounding air to such an extent that its bulk will increase four times in twenty-four hours.

A fuming acid (Nordhausen sulphuric acid), $H_2S_2O_7$, is extensively used in the arts. This fuming acid is the product of the distillation of ferrous sulphate in the dry state at a nearly white heat; the distillate is received in sulphuric acid. This dark-colored, fuming

acid has a specific gravity of 1.9, and boils at 53° C. This acid is especially used as a solvent for a dye called alizarin.

Sulphuric acid is used in the manufacture of other acids and chemical substances, as, also, in making alkalies, in the manufacture of indigo and other dyes, in the making of soaps, artificial manure, and for many other industrial purposes.

78. Local action; symptoms of poisoning.—The local action of sulphuric acid, as given by Mr. Blyth,[1] who refers to the experiments of Falck and Victor on living animal tissues, in its concentrated strength, will precipitate albumen, and afterwards dissolve it; fibrin swells and becomes gelatinous; if the acid be dilute (4 to 6 per cent), fibrin is scarcely changed. Muscular tissue is at first colored amber-brown, swells to a jelly, and then dissolves into a red-brown, turbid fluid. When the acid is placed in contact with the mucosa of the stomach, this tissue, as well as the layers of muscle beneath it, are colored white, swell, and become an oily mass.

"When applied to a rabbit's ear, the parenchyma becomes at first pale grey and semitransparent at the back of the ear; opposite the drop of acid appear spots like grease or fat-drops, which soon coalesce. The epidermis with the hair remains adherent; the blood vessels are narrowed in caliber, and the blood, first in the veins and then in the arteries, is colored green and then black, and fully coagulates. If the drop, with horizontal holding of the ear, is dried in, an inflammatory zone surrounds the burnt spot, in which the blood circulates; but there is complete stasis in the part to which the acid has been applied. If the point of the ear is dipped in the acid, the cauterized part rolls inwards; after the lapse of eighteen hours the part is brown and parchment-like, with scattered points of coagulated blood; then there is a slight swelling in the healthy tissues, and a small zone of redness; within fourteen days a bladder-like, greenish-yellow scab is formed, the burnt part itself remaining dry. The vessels from the surrounding zone of redness gradually penetrate towards the cauterized spot, the fluid in the bleb becomes absorbed, and the destroyed tissues fall off in the form of a crust.

"The changes that sulphuric acid causes in the blood are as follows: The fibrin is at first coagulated and dissolved, and the coloring matter becomes black. These changes do not require the strongest acid, being seen with an acid of 60 per cent."

It therefore follows from the above action of sulphuric acid on the

[1] Poisons: Their Effects and Detection, 3d ed. op. cit.

living tissues, as well as that of the other stronger mineral acids, that it will produce, when brought in contact with the human tissues, a corrosive or caustic action, and this explains why they act as a poison to endanger or destroy life.

Sulphuric acid is more frequently taken and given by mistake than by design, at least in our country, although cases are not wanting in which it has been criminally given to young children. An interesting case is reported in Henke's Zeitschrift, where it was used with the hope of inducing abortion. From the ease with which it can be procured, without awakening suspicion, on account of its extensive use in the arts, either pure or mixed with other substances, it has frequently been taken with suicidal intent; but on account of the excessive pain produced by this poison, and on account of the facility with which other less painful means of procuring death can be obtained, the frequency of sulphuric acid poisoning has much diminished of late years,—especially in this country and France. Most of the cases seen in recent years have been cases of accidental poisoning; it has, for instance, been taken by mistake for castor oil. Very rarely is sulphuric acid administered with criminal intent, its intense acidity, and the immediate pain caused by it, enabling the intended victim to detect it at once. It can only be employed for the purpose of murder in the case of young children or drunken persons, unless very great force be used, which would require the presence of several criminals. This acid has also proved fatal when used as an injection into the vagina for the purpose of procuring an abortion,[2] and when injected into the rectum either accidentally or designedly;[3] and of late years, in England and the United States, numerous cases have occurred where it has been employed for disfiguring the person, by being clandestinely thrown upon the face or neck.

Sulphuric acid may be taken as the type of the corrosive poisons. The symptoms, of course, vary very greatly according to the concentration and the quantity of the acid taken. When a large dose of the concentrated acid has been taken, the symptoms begin almost immediately, and are characterized by a very acute pain, which at first is that of burning, and extends from the mouth to the stomach. This abdominal pain is most excruciating, somewhat resembling that of cholera, and is located by the patient chiefly in the epigastrium. Violent and painful vomiting also occurs almost immediately; the

[2] Jour. de Chim. Médic. tome VII. 1831, Mai, No. 10, p. 312.

[3] Preuss. Med. Vereins-Zeitung, 1848, No. 13, where the acid was used by mistake for oil; and Hofmann, Lehrbuch der gericht. Medicin, 1878, p. 634, where the acid may have been used intentionally.

vomitus consisting usually of black or dark brown material (so-called coffee-ground vomitus), of tarry consistency, and very acid in its reaction, so that if it happens to fall upon a marble slab or other calcareous material, effervescence will take place. The dark brown or black vomitus is often frothy, and mingled with shreds of stained mucous membrane. The character of the contents of the stomach will have an important bearing upon the period at which death occurs, as well as upon the amount of destruction of the tissue of that organ which is exposed to the action of the acid, except in those cases in which a large amount of acid is swallowed. For instance, if the contents were composed largely of milk and albuminous food, of fats and oleaginous substances, they might offer some protection to the walls of the stomach, which latter might escape serious injury. These foregoing remarks explain the wide range of time in which fatal results occur in persons who have taken about the same dose, due to the action of the acid upon the blood. The microscopic examination of the vomitus shows the presence of gastric cells, and later, shreds of tissue from the walls of the stomach, sometimes even tubular bodies consisting of the exfoliated membrane of the œsophagus, are seen. This vomiting usually takes place immediately after the swallowing of the acid, but there are a few cases in which vomiting was delayed for some time; the condition of the stomach, whether full or empty, will explain most of these cases of delayed vomiting.

In some cases the concentrated acid comes in contact with the larynx, or with the mucous membrane of the air passages, when it may cause edema of the glottis, and death in a few minutes from suffocation.

Evidences of asphyxia will be seen at the post-mortem examination. This is especially liable to be the case when the sulphuric acid has been forcibly administered, as, for instance, to infants. Sometimes sudden death is due to perforation of the stomach or to extensive hemorrhage, due to the opening of one or more of the large blood vessels by the corrosive action of the acid.

In ordinary cases where life is prolonged for several hours, the average duration being from eighteen to twenty-four hours, collapse takes place in a short time. There is almost complete suppression of urine, due to irritation of the kidneys caused by the acid; absence of feces, although we occasionally see diarrhea; the pulse is weak, the skin is cold and clammy, and the face is expressive of the most intense anguish; usually the intellect remains intact until death occurs. Sometimes convulsions are present, which are similar to those often

seen in cholera. Rarely an intermission occurs in the symptoms, as in the case of many of the irritant poisons, shortly followed by an exacerbation and death.

Unless care be taken in administering or taking the acid, we can usually see evidences of its local action upon the skin of the face or neck, or upon the mucous membrane of the mouth and lips, and, in many cases, stains are made by the acid upon the clothing. The eschars produced upon the skin and mucous membranes are at first white, changing to a gray, and finally to a brown or black color.

The peculiar symptoms are the great suddenness of the attack, the extreme pain, the character of the stains upon the mucous membranes, skin, and clothing, the constipation, and the anguish which precedes death. The localization of the pain in the epigastrium is also an important symptom from a diagnostic point of view, since, in the case of many of the other irritants, the pain is more diffused over the entire abdominal region.

79. Subacute poisoning.— When the dose of sulphuric acid has been small, or taken upon a full stomach, or when the acid has been quite dilute, the symptoms may be less severe, and produce the effect of subacute poisoning. In these cases life may be prolonged for months or even years, the patient finally dying of marasmus, consequent upon the incurable disturbance of the digestive organs due to the partial destruction of the mucous membrane of the stomach. At first, in these cases, the symptoms resemble those of acute poisoning, but are much less severe; there may or may not be hemorrhage, as shown by bloody vomitus; the vomitus is usually glairy, containing much mucus; there is excessive pain in the mouth and throat, this pain extending to the stomach, and rendering the ingestion of food almost impossible. After the first eighteen or twenty-four hours, in either acute or subacute poisoning, inflammatory symptoms appear, due to the inflammation of those tissues with which the acid has been in contact; these symptoms are those of febrile reaction, accompanied with rapid pulse, rise of temperature, etc. Soon, shreds of the disorganized membrane may be expelled by vomiting, and if the ulcerative process has affected the walls of the larger blood vessels, there may be hemorrhage. If the patient's life be spared for a sufficiently long time for the ulcerations caused by the sulphuric acid to heal, the cicatrices resulting may cause stricture of the œsophagus, stomach, or intestines, with the long train of symptoms due to the consequent obstruction, dyspepsia, and indigestion, until finally, after perhaps months or years of suffering, the patient dies from inanition.

Though in subacute poisoning from sulphuric acid there are usually secondary inflammatory processes, the results of which are contracted cicatricial adhesions of the mucous and subjacent tissues, there is associated with this condition an enfeebled digestion of food, and stricture of the gullet, as well as, sometimes, stricture of the entrance and exit passages of the stomach, which are difficult to relieve. Mannkopf has recorded a curious sequel of an obstinate intercostal neuralgia, which he observed on the fourth, seventh, and twenty-second day after the first symptoms of poisoning.

80. Chronic poisoning.—It will be observed from the secondary effects which we have above related, that, even after the acute and subacute poisonous symptoms have disappeared, a chronic poisonous condition may result from the alteration in the normal condition of the circulating blood in the tissues of the human body. H. Weiske[4] administered to lambs small doses of sulphuric acid, and observed, as a result, that they became emaciated from this treatment, and that their smaller bones showed a diminution of lime salts, as did also their muscles. Kobert[5] offers his opinion that a continued use of "Schnapps," commonly drunk on the continent, shows typical signs of chronic sulphuric acid poisoning; this drink is made up with sulphuric acid to give it a sharp taste. Mr. Blyth[6] presents a typical case of chronic poisoning by this agent, which is reported by Oscar Wyss: "A cook, thirty-four years of age, who had suffered many ailments, drank, on the 6th of November, 1867, by mistake, two mouthfuls of a mixture of 1 part sulphuric acid and 4 of water. Pain in the stomach and neck, and vomiting of black masses, were the immediate symptoms, and two hours later he was admitted into the hospital in a state of collapse, with cold extremities, cyanosis of the face, etc. Copious draughts of milk were given, and the patient vomited much, the vomit still continuing of black pultaceous matters, in which, on a microscopical examination, could readily be detected columnar epithelium of the stomach and mucous tissue elements. The urine was of specific gravity 1.033, nonalbuminous; on analysis it contained 3.388 grams of combined sulphuric acid.

"On the second day there was some improvement in the symptoms, the urine contained 1.276 grams of combined sulphuric acid; on the third day 2.665 grams of combined sulphuric acid; and on the tenth day the patient vomited up a complete cast of the mucous membrane of the gullet. The patient remained in the hospital, and became

[4] Jour. f. Landwirthsch. 1887, p. 417. [6] Op. cit. p. 84.
[5] Lehrbuch der Intoxicationen, s. 210.

gradually weaker from stricture of the gullet and impairment of the digestive powers, and died two months after taking the poison, on the 5th of January." The writer is familiar with a case in which an ounce of sulphuric acid (strength unknown) was taken by mistake for cider; the patient recovered from the acute symptoms, but died a little more than two years after, on account of the secondary troubles in the digestive organs, which have been alluded to above.

81. Fatal dose.— It would be impossible to offer an opinion as to what constitutes a fatal dose. An opinion would be much influenced by a variety of circumstances arising in each case. Taylor records one case in which 20 drops of concentrated sulphuric acid caused death in a child twelve months old. Christison records a case of an adult who died from the effects of a dose of 3.8 grams (60 gr.) which is the smallest lethal dose known to have been fatal. The varying circumstances affecting the size of a fatal dose depend upon the strength of the acid administered, on the fact whether the stomach is filled with food, upon which a portion of the acid would be expended, whether the stomach contains a large amount of water or milk or demulcent, such as oil, etc., and, on the other hand, whether the stomach is completely empty. The local action on the mouth and gullet would probably be the same, when compared with similar strength of the acid ingested. It is always difficult, however, to ascertain the exact quantity which has been swallowed, since the immediate impression made upon the mouth by the contact of so corrosive a liquid naturally excites an instantaneous effort to get rid of it. The amount actually swallowed forms usually, therefore, but a small portion of what is taken into the mouth. It may, indeed, not reach the stomach at all, its action being expended upon the throat and œsophagus, and proving fatal by the inflammation and disorganization there produced.[7] Occasionally, also, it enters the air passages. A case of recovery after an injection of sulphuric acid, given by mistake for castor oil, is reported.[8]

There are many recorded cases of sulphuric acid poisoning; the shortest periods, after swallowing the acid, before death has occurred, vary from a half to two and a half hours. The rapidity of the death will depend upon the character and extent of the primary lesion; for instance, if a perforation occurs in the stomach or intestines, death will rapidly follow from the consequent collapse. The

[7] This happened in a case reported by Dr. W. Hull, in the Lond. Med. Gazette, June, 1850.

[8] Ed. Month. Jour. April, 1850.

tardy appearance of death will be influenced by the character of the tissue destroyed or organs impaired; for instance, if the secondary results from the destruction of tissue in the region of the throat cause inflammatory processes and consequent contractions, by which the passage through the air tubes or the gullet is obstructed, loss of life may occur from interference with the organs of respiration, or from starvation from an obstruction to the ingestion of food. Sometimes the limits of these inflammatory lesions have increased gradually in extent during weeks and months, and thus death has been indirectly caused at a period of time far remote from the moment when the acid was swallowed. The smallest quantity which has been taken with rapidly fatal results appears to be that which was given in a case quoted by Dr. Taylor. In it, the quantity is said not to have exceeded 40 drops. But the patient was a child only a year old, and antidotes were administered about a half hour after it had taken the acid.

82. **Fatal period.**—Usually death takes place within a day, or during the succeeding twelve hours; but a case of death occurring within an hour is recorded, after a dose of 3½ ounces by measure (103 c.c.) of the concentrated acid. This reservation should be made, however. It may happen that the contact of the corrosive may be held long enough in the mouth and throat to injure these local tissues, and produce an edema or swelling of these parts so as to prevent air passing into the lungs; this would cause death by suffocation.

In subacute cases life may be prolonged for a week or two. In chronic cases it may be prolonged for months or years, and death occur from serious injury to the surface of the digestive tract, on account of the destruction of the glandular cells contained in the mucosa and its subjacent tissues. In these cases of prolonged action we should expect the result would be preceded by symptoms of lack of nourishment of the general body, and end in inanition. It should also be remembered that the presence of acid in the blood interferes with the normal processes of metabolism within the tissues throughout the body, which are concerned with the repair and construction of healthy tissue formation, and this condition may also interfere with the normal functions of such organs as the liver, spleen, kidneys, etc. There is, besides the case previously alluded to, one case on record, referred to by Beck,[9] in which death, the result of stricture of the œsophagus, did not occur until two years after the poison had been taken.

[9] See Taylor on Poisons.

83. Diagnosis.— The local action of corrosives is so apparent to the physician of ordinary experience that he should be able to recognize a case of poisoning from any one of them, but to determine which of the corrosives has been ingested should be learned, apart from the character of the caustic action which has been previously described, by the test of litmus solution or litmus paper, as well as the action of the vomited matter upon vegetable or animal substances upon which it may come in contact. Wood almost immediately blackens, and the spot remains moist; when the acid falls on vegetable earth, effervescence occurs from the decomposition of the carbonates; grass or vegetation blackens and dies; where the acid falls on cotton fabric, a black spot will be left, and probably a hole; when acid falls on colored cloth, the spot will become red or brown, and will be moist. Indigo-blue dyes will not be stained red; logwood and madder dyes will be stained yellow.

84. Treatment.— After discovering that we have a case of poisoning from an acid corrosive, effort should first be made to neutralize the acid by means of chalk, magnesia, or a solution of sodic carbonate in a large amount of water; it is suggested that the sooner the acid is neutralized the better, and, if none of these articles are at hand, plaster scraped from the ceiling or wall may be utilized as a neutralizing agent. Plenty of water should be freely used; after the neutralizing of the acid swallowed has been accomplished, demulcent and oily drinks or fats should be administered, and, to prevent suffocation from the closing up of the entrance to the air-passage, the operation of tracheotomy may be indicated.

85. Pathology of sulphuric acid poisoning.—The pathological effects of poisoning by sulphuric acid, though this poison is less frequently used in this country and Europe to destroy life, show black eschars in the stomach, and the stomach contents are observed to be black and of a tarry consistency, but sometimes more fluid. These eschars, if examined soon after death, are somewhat hard and brittle, and then become easily separated and stripped off from the underlying tissue. Phagedenic inflammation accompanied by small hemorrhagic points may be noticed, and the destructive action of this acid may eat through animal tissues and cause perforations.

The corrosive action of this acid is frequently observed upon the outside of the lips and on the skin of the face, and this corrosion may extend inside the mouth and throat, both into and involving the entrance to the air passages and to the gullet; its corrosive action may even be observed beyond the stomach,—in the intestines. The blood

is thick and clotted; the parenchyma of the liver, kidneys, and even the heart may be seen to show a degeneration. Though death may be caused by the destruction of the local tissues from the direct corrosive action due to the presence of the acid in contact with them, yet, as death has been observed to follow the ingestion of the dilute sulphuric acid,—from the contact of which we should expect this action to be very feeble on the local tisues,—it is believed that the lethal action may be brought about by its presence in the blood, neutralizing the alkali which is necessary to retain its fluid consistency. The following case will illustrate the effects of this poison when concentrated and swallowed: A man thirty years of age went to his closet in the dark, and drank a "good mouthful" of commercial sulphuric acid. He was bled shortly after, and the blood is described as having a syrupy consistence. Milk and soapsuds were given to him, followed by vomiting; but he died in two hours. The whole tongue was white and sphacelated, and the mucous membrane here and there detached. The fauces and œsophagus were of a grayish-black color; the stomach was perfectly black on both surfaces, and of a soft and pulpy consistence. The omentum majus was likewise partly carbonized, in consequence of the escape of the acid into the abdominal cavity. The upper portion of the small intestine was of a blackish color, and the mucous membrane swollen and indurated. The contents of the stomach yielded, on chemical examination, 5 grams (77.25 gr.) of free sulphuric acid.[10] In Mr. Traill's case, a washerwoman took a wineglassful of crude commercial acid of the specific gravity of 1.833, by mistake for ale. Although the proper antidotes were very soon administered, she died in one hour. A hole with ragged edges was found at the fundus of the stomach, and the adjacent tissue tore with the slightest touch. The rest of the mucous membrane was mottled with dark-brown patches. There was also great inflammation of the peritoneum, from the escape of the acid.[11] When much acid has been swallowed, the stomach presents an appearance which is altogether peculiar and characteristic. It is as black as coal, and its tissue is softened to a jelly. The acid, softening the walls of the blood vessels, allows their contents to escape, and then, acting upon the blood, gives it a dark color. That such is the source of the black color referred to is proved by the fact that it is not produced when sulphuric acid is introduced into a dead stomach.[12]

[10] Casper, Gericht. Leichenöff. 1 H. p.

[11] Month. Jour. Aug. 1854.

[12] Orfila has established the fact that this acid, even when taken in a concentrated form, can be absorbed after it has been diluted in the stomach and intestinal canal and united with liquids therein contained, or which have afterwards

In general, the eroded and inflamed appearance of the mouth and throat is found to coexist with the blackened and disorganized condition of the stomach. But sometimes the poison does not reach the stomach, and, when this is the case with young children, death may take place from the local action on the fauces alone. In Dr. Hull's case, already referred to, it reached the lungs. The epiglottis was partially destroyed, the vocal cords and the mucous membrane of the trachea were softened, the left pleura was perforated, and a crust of sulphate of calcium formed upon the ribs. There was no trace of the acid, either in the œsophagus or stomach. The person was an adult female. It is probable, in such cases, that death takes place by suffocation on account of edema of the glottis. Husemann reports two fatal cases in children of five years of age. The symptoms were chiefly those of inflammation of the throat and larynx, without any evidence of disorder of the stomach.[13] No autopsy was made. In most cases of poisoning with sulphuric acid, there are also traces of its action left upon the skin, near the mouth, either from a portion escaping when swallowed, or from the corrosion of the vomited liquids. The marks thus left upon the skin are of a dark brown color, and of a leathery consistence. Where, however, the acid has been given in a spoon, the anterior portion of the mouth may escape entirely. In a case referred to by Dr. Taylor, the fauces, œsophagus, and stomach of an infant ten days old were much corroded by sulphuric acid, but there was no appearance of injury to the mouth. This was probably owing to a spoon having been used, and the poison having been poured down the throat slowly, as the mucous membrane was extensively corroded at the back part. A case is reported, in which, although the acid was taken from a teacup to the amount of 15½ drams, there was not the slightest vestige of a stain on the outside of the lips, angles of the mouth, cheeks, neck, or hands, nor upon the clothing.[14]

There is a case on record[15] where a woman swallowed from a cup about a tablespoonful of strong sulphuric acid; the usual symptoms appeared, and she died in two days. The mucous membrane of the cheeks, gums, and tongue was not excoriated at any part.

We find a case of gelatiniform softening of the stomach, with per-

been swallowed; and that, consequently, in autopsies, all the viscera and the urine should be examined for the presence of this acid, even though nothing is found either in the alimentary canal, vomited matters, or the feces.

[13] Jour. f. Pharmakodyn. II. 166.

[14] Dr. Walker, Ed. Month. Jour. June, 1850.

[15] Dr. Chowne, Lancet, July 10, 1847, p. 36.

foration of its fundus, and effusion of a brownish liquid into the peritoneal cavity, reported by Dr. Casper. In this case there had existed suspicion of poisoning, which his opinion from the result of the examination set at rest. Were it admitted, in the absence of direct proof by chemical analysis of the contents of the stomach, that an inflammatory condition, a softening, or ulceration of the stomach could be regarded, apart from any corrosion of the mouth or fauces, as presumptive evidence of poisoning, we should be led, undoubtedly, into many grave mistakes. Spontaneous softening of the stomach in infants, and ulceration and perforation of this organ in adults, are not rare events, but their characters are far different from the charring and disorganization produced by sulphuric acid. We think that in no case is an opinion warranted that sulphuric acid has been swallowed, unless it can be clearly traced by its effects from the mouth or fauces to the stomach.

Dr. Norman Moore[16] reported the post-mortem appearances in a case of poisoning of a man, who lived only one and a half hours after swallowing ½ pint of the oil of vitriol of commerce. As this report was carefully made, and because there was evidence that the poison corroded the tissues after death, as well as before, we present it in detail. The mucous membrane of tongue and fauces was grayish, not charred. The walls of the gullet were soaked with the acid, which had even diffused into the pericardium and posterior walls of the heart. Neither gullet nor diaphragm was perforated. One eighth of an inch in thickness of the bottom of the lung substance was hardened by imbibition of the acid. The stomach and upper portions of the small intestine were quite rotten from the action of the acid; the lower portions of the small intestine, and also of the large intestine as far as the rectum, were grayish and evidently had been attacked by the acid. The surface of the liver was also hardened, and all of the abdominal viscera were more or less acted upon by the corrosive, and the saturation had even affected the outside wall of the abdomen; the neighborhood of the navel being grayish also. Many of these changes were undoubtedly, in the opinion of Dr. Moore, due to the action of the acid upon the tissue after death.

It has already been mentioned that in a case of poisoning by this acid the blood had a syrupy consistence. This condition is stated by Casper to be the ordinary one. He adds that the blood is generally of a cherry-red color, and that it has an acid reaction. This acidity

[16] Brit. Med. Jour. London, 1879 Pt. I. p. 430.

has also been noticed in a pericardial effusion, and in the amniotic liquid of a pregnant female poisoned by sulphuric acid. It would appear that after death by this poison bodies remain fresh and without smell for an unusually long time; a fact which Casper explains by stating that the acid continues, until exhausted, to neutralize the ammonia which is given off in decomposition.

We also sometimes see the inflammatory action of the acid upon the organs, due to its having been absorbed and carried to them through the circulation; thus, the kidneys are almost always irritated by the acid, sometimes to such an extent that bloody urine is secreted by them, and the urine is almost always albuminous and during life gives evidence of this irritation of the kidneys; Mannkopf in one of his cases[17] mentions having found a fatty liver, as in the case of many of the other irritant poisons.

In cases of subacute poisoning when life has been sufficiently prolonged, we always find the results of the previous destruction of tissue, in the form of cicatricial contractions or strictures of the œsophagus, stomach, intestines, or trachea, and hypertrophy of the walls of these organs, wherever they were attacked by the acid; also we frequently see ulcerations, which either have never healed or have been produced secondarily.

86. Poisoning by ink.— A drunken soldier had given to him a large glass of ink, under the pretense that it was porter. He drank it, and, after sleeping for an hour, awoke in the most violent pain. He suffered extreme weakness, headache, and painful cramps in the thighs. After four or five hours he commenced vomiting a pasty mass mixed with ink, which gave strong indications of sulphuric acid. Mucilaginous and saccharine beverages were given him, and after a short time he improved. He was convalescent on the third day, but still complained of weakness, trembling, and an oppressive pain in the back of the head.[18] The presence of sulphuric acid in the ink is explained by the decomposition occurring between the sulphate of iron and tannic acid, of which some inks are made.

87. Tests.— Inasmuch as sulphates constitute a portion of our food, the presence of them is no indication of poisoning by sulphuric acid. On the other hand, the administration of antidotes may render the stomach contents neutral or even alkaline, and no free sulphuric acid be detectable, or vomiting may have been so severe that practically all the acid has been expelled. In any case, as Buchner well says, "the

[17] Wien Med. Wochensch. 1862, No. 35, and 1863, No. 5.

[18] Am. Jour. Med. Sci. April, 1854, from Pharmaceutical Jour. Oct. 1853.

chemical detection of poisoning by nitric or sulphuric acid is as a rule impossible." It is therefore necessary, in a case suspected to be one of sulphuric acid poisoning, to resort to tests not commonly employed in other cases. The ordinary test for sulphuric acid or sulphates is the addition of barium chlorid and hydrochloric acid, producing a precipitate of barium sulphate. The addition of the hydrochloric acid is to prevent the precipitation of barium carbonate, phosphate, etc., yet the HCl must not be added in excess, as barium chlorid itself is insoluble in concentrated HCl.

Further tests must be made for free sulphuric acid. Litmus shows an acid reaction due to free acid, acid salts, or organic acids, so cannot be used as a test for a free acid. Certain anilin dyes, such as congo red, react to mineral acids, but not in the same way as they do to acid salts or organic acids; with the former it gives a dark blue color, with the latter a violet; an alcoholic solution of tropaeolin with a drop of mineral acid evaporated on a bit of porcelain gives a brown-red spot turning violet; Gunzburg's reagent (phloroglucin vanillin) gives a beautiful red when gently evaporated on porcelain with a few drops of a mineral acid.

It is often necessary—especially where tests for free sulphuric acid have proved negative—to make a quantitative estimation of the amount of sulphate present, and, if it is largely in excess of the normal, it forms a presumption in favor of the presence of free sulphuric acid.

In view of this, and the fact that the weight of the evidence of the quantitative estimation, unless the latter be excessive, is of doubtful value, the chief evidence must be obtained rather from symptoms during life and the post-mortem investigation than from a chemical analysis.

88. Stains on clothing.— The stains on blue and black cloth, made by dilute sulphuric acid, are at first red, and afterwards brown, and the stuff is corroded. Concentrated acid produces a brown stain at once, owing to the destruction of the fibre and the charring of the material. When a drop of dilute acid falls upon cloth, a portion of the water gradually evaporates, until the acid reaches a certain degree of concentration; hence the change in the color of the stain from red to brown. The color of black leather is not altered, but the substance is partially destroyed. The stains on all textile fabrics remain moist for a considerable time if the acid has been strong, and, owing to the attraction of the acid for water, they have an unctuous feeling. The acid may be detected in these stains after the lapse of many years.

Dr. Taylor has detected it in spots made upon a black cloth dress twenty-seven years previously. The stains should be cut out, digested in distilled water, and then the liquid, after filtration, be tested by the reagents already mentioned. An unstained portion of the cloth should be submitted to the same test, since many articles of clothing yield slight traces of sulphates when boiled in water.

89. Sulphate of indigo.—This is a solution of indigo in Nordhausen, or fuming, sulphuric acid. In addition to the other symptoms of poisoning by a corrosive liquid, the deep blue and subsequently greenish color of the vomited matters will at once betray the agent that has been used. In some cases the urine has had a bluish tinge.

II. Hydrochloric acid.

90. In general.— Hydrochloric acid is largely used in the arts and manufactures, in bleaching processes, etc., and also in medicine. It is a colorless gas (sp. gr. 1.26), very soluble in water; about 450 volumes HCl dissolve in 1 volume H_2O; acid taste and reaction.

The ordinary form in which we meet with hydrochloric acid is as an aqueous solution of the gas, of varying strength and purity. It is often known as muriatic acid or spirit of salt. The ordinary commercial acid is generally colored more or less yellow from an impurity of ferric chlorid, and also contains traces of sodium and arsenic, as well as free chlorin and sulphur dioxid. It contains about 32 per cent HCl. The HCl of the U. S. P. is a colorless liquid containing 31.9 per cent HCl and only traces of impurities. The dilute U. S. P. acid contains 10 per cent of HCl by weight.

On exposure to air the strong acid gives off fumes which are distinctly visible, and which, if they come in contact with ammonia, form dense clouds of ammonium chlorid. The acid attacks most metals, forming the various chlorids, and evolving hydrogen. It also dissolves most of the basic oxids and hydrates and carbonates with the formation of CO_2.

91. Acute symptoms.— The reported cases of poisoning with this acid are few in number; but they present a strong analogy in their symptoms and post-mortem appearances with those of sulphuric acid poisoning. It should be remarked that, owing to its volatility, this acid, by the irritating fumes set free in the warmer atmosphere within the mouth, may set up an inflammatory action upon the glottis and throat; and though the liquid acid itself may not come in contact

with the laryngeal tissue, in such cases we may find conditions in these tissues, which may produce symptoms of difficult respiration. There have been recorded only a few cases of murder by this poisonous acid, and these chiefly in infants; it is easy to understand the difficulty of administering to an adult so strong and repulsive an agent. Immediately upon swallowing it there is a burning sensation from the mouth to the stomach, but especially in the throat, attended with a feeling of suffocation, and followed by uncontrollable vomiting. Deglutition is almost impossible, all efforts to swallow bringing on vomiting; the voice is low and the respiration frequent and labored. The tongue and fauces are usually covered at first with a whitish pellicle, which afterwards becomes detached, exhibiting corroded spots underneath, and later becoming brown and pultaceous. Then there follows a feeble pulse and exhaustion, though the intellectual faculties may remain clear to the last. These acute symptoms may cause a fatal issue within an hour or two,—especially if the entrance to the larynx and air passages has been seriously attacked by the volatile fumes,—and produce an edematous swelling which may impede the inspiration of air to the lungs; or, in absence of this local action, life may be prolonged twelve or twenty-four hours. Sometimes, but rarely, death has occurred from a perforation of the stomach.

92. Subacute poisoning.—If the victim should survive the immediate and threatening symptoms above described, which are similar to those in the case of poisoning by the other strong mineral acids, the secondary inflammation, mentioned as also being liable to occur, may produce stricture or occlusion of the passages in the throat, trachea, and gullet, which may supervene and interfere with the functions of breathing and primary digestion of food. Life may be prolonged a few days, but under very adverse conditions, and will terminate slowly but surely by gradual exhaustion or from deficient nutrition.

93. Chronic poisoning.— If the symptoms above related are not followed by death within the first week, the patient may suffer from the effects of the impairment of the general health from disturbances of the digestive tract; and from a train of symptoms we have described in the instance of the other mineral acids, from the destruction of the functions of the lining membranes and subjacent tissues in the general alimentary tract, and, also, from changing the normal alkaline condition of the blood, which involves the interchanges in the assimilative processes of the human tissue, from this alteration of the physico-chemical character of the blood. In a case observed by M.

Guérard,[19] a woman aged twenty-four years, who had swallowed about 53 grains of concentrated hydrochloric acid, presented the above symptoms. She, however, survived a considerable time. The matters vomited on the second night did not present any acid reaction, although no chemical antidote appears to have been administered. Death took place two months after the poison had been swallowed; and some time previous, portions of corroded mucous membrane were discharged both by vomiting and by stool. Profuse salivation also was observed in this case; and in the beginning white vapors were exhaled from the mouth. The same symptoms and the same prolongation of life were noticed in a case which became the subject of judicial investigation in Belgium, and which is commented upon by Orfila.[20] In two cases referred to by Dr. Christison, and in another, of a child, by Orfila,[21] death took place within twenty-four hours. In this latter instance, the acid was poured down the child's throat by its stepmother, as confessed by her after her condemnation. A case of recovery after swallowing 1 ounce of strong hydrochloric acid is reported in the Lancet for 1850. In this case the stomach pump was used, contrary to the usual caution enjoined in poisoning by mineral acids.

94. Diagnosis.— The only means of recognizing poisoning by hydrochloric acid, in contradistinction from the preceding acid, would be by a differential diagnosis; for instance, if the contents of the stomach or mouth containing this acid should drop upon a cloth, a reddish spot would appear, only to disappear shortly, because hydrochloric acid is volatile; this would be the same with the appearance of black dyed cloth, which is sometimes turned to a greenish stain, but these spots are not moist or charred, as by sulphuric acid, or rotten; nor are they yellow as by the action of nitric acid.

95. Treatment.— The same method for the relief of poisoning by hydrocholoric acid is recommended as with sulphuric acid (see § 84, *supra*).

96. Post-mortem appearances.— These vary according to the length of time the person has survived, but bear a general resemblance to the effects produced by the other strong mineral acids. The digestive mucous membrane is covered with whitish, superficial sloughs, which subsequently become of a darker color, and are found in all stages, lying loose or partly detached, and the mucous membrane inflamed, softened, or corroded. In some of the above cases, all the coats of

[19] Ann. d'Hygiène, tome XLVIII. p. 415.

[20] *Ibid.* tome XI. p. 35.

[21] *Ibid.*

the stomach were destroyed in spots, and perforations resulted. In Guérard's case, there was slight peritonitis. It is important, however, to note that the peritonitis resulting from perforation of the stomach only occurred in those cases which survived a long time. In Dr. Budd's case the force of the poison was spent upon the mouth, fauces, and larynx. The mucous membrane of these parts was whitened and softened, the soft palate and tonsils swollen, and a portion of the lining membrane of the larynx was entirely removed.

Schmaus and Ewing[21a] mention that "nitric and hydrochloric acids have about the same (pathological) effect; but the former causes an orange-yellow color (xanthoprotein) where concentrated, and a violet to grayish white where more dilute, and the latter produces a grayish-yellow color."

97. Tests.— Silver nitrate produces a white curdy precipitate of silver chlorid, soluble in ammonium hydrate or potassium cyanid solution, insoluble in nitric acid.

Mercurous nitrate produces a white precipitate of mercurous chlorid, calomel, which is blackened by the addition of ammonium hydrate.

Lead acetate produces a white precipitate in fairly strong solutions of chlorids. This lead chlorid is only slightly soluble in cold water, quite freely so in hot water, and may be crystallized from the latter in long needles.

When warmed with manganese dioxid, free hydrochloric acid evolves chlorin, which may be recognized by its color if in large quantity, also by its odor and bleaching power on moist vegetable coloring matters.

The following means of detecting hydrochloric acid in cases of suspected poisoning has been suggested (J. Bouis, Journal of Applied Chemistry): Filter the liquid through linen and paper previously moistened with acetic acid, and then introduce a few crystals of chlorate of potash and some gold foil. Heat the whole over a water bath an hour or more, and the presence of the free acid is determined by the quantity of gold dissolved by the liquid. Chlorid of tin may be used to detect even a few faint traces of gold which may have been dissolved. Diluted liquids should, before applying the above process, be concentrated by evaporation. A very few centigrams of hydrochloric acid can be detected in a large quantity of liquid. The chlorids of sodium, potassium, and the like do not produce similar reactions.

21a Op. cit. p. 362.

The detection of hydrochloric acid in the stomach must be ascertained quantitatively in all cases of suspected HCl poisoning. The presence normally of HCl to the extent of 0.2 per cent, more or less, renders this quantitative determination essential. Further, it is not sufficient to determine the amount of free HCl in the stomach, but it must be taken into consideration that some of the HCl has combined with the proteid of the food, and is then in the form of an organic salt or combined salt.

One simple method for this is as follows: An aliquot part of finely divided material is evaporated to dryness, and the amount of Cl determined with silver nitrate solution. Another aliquot portion is first neutralized with sodium carbonate, and then treated as the other. If, now, this latter portion shows more chlorin than the first, the excess corresponds to the amount of free hydrochloric acid in that portion.

As common salt is almost invariably present in the stomach, and is a natural constituent of most of the secretions, the detection of HCl forms no indication of the manner in which it was introduced.

III. Nitric acid.

98. In general.—Nitric acid (HNO_3) is also known as aqua fortis, chemically as nitric acid, hydric nitrate, or nitric monohydrate.

Concentrated nitric acid, as met with in commerce, varies in color from a light yellow to a deep orange-red, owing to the presence of nitrous or hyponitrous acid, but the pure acid is colorless. Red fumes of nitrous acid gas are given off when a few copper filings are treated with nitric acid. It produces a yellow stain upon the skin, mucous membranes, or cloth, which changes to an orange upon being neutralized with an alkali; this yellow color is due to the action of the nitric acid upon albuminous matters, with which it forms xanthoproteic acid. Poisoning by nitric acid is much rarer than by sulphuric acid, since the odor of the fumes evolved from the strong solutions permits its ready detection before any of the acid has been taken into the mouth; hence a case of accidental poisoning by this acid is exceedingly rare, although it is very extensively used in the arts. The fumes arising from concentrated nitric acid are also poisonous when inhaled, and fatal cases have been recorded from the inhalation of nitrous fumes in laboratories and factories.

99. Preparations.—The officinal preparations (U. S. P.) are acidum nitricum dilutum. Acidum nitrohydrochloricum. Acidum nitro-

hydrochloricum dilutum. The dilute nitric acid contains 1 part of pure acid to 6 parts of water. Nitrohydrochloric acid contains 4 parts of nitric acid to 15 parts of hydrochloric acid. Dilute nitrohydrochloric acid contains 4 parts of nitric acid to 15 parts of hydrochloric acid and 76 parts of water.

100. Symptoms of poisoning.— When concentrated nitric acid is in contact with the skin, it fumes and small bubbles of gas are evolved; the surface of the skin will soon be stained yellow, and the stains will not disappear by the addition of ammonia; the yellow tinge will be changed into a reddish-orange color on being moistened with potash and a solution of potassium cyanid. When this acid comes in contact with the mucous membrane of the mouth, there may be observed a similar yellowish stain, of a lighter shade, however, than the stain on the skin; on the mucous membrane of the gullet and stomach there is the usual staining and, in addition, yellow and black eschars, also pulpy shreds of the mucous and subjacent tissue; there are rare instances of perforation. The corrosive action produced by swallowing strong nitric acid is followed by symptoms which do not differ essentially from those which have already been mentioned as caused by sulphuric acid. There is the same intense, burning pain in the mouth, throat, and stomach, the same violent efforts to vomit, and urgent constitutional symptoms. The lining membrane of the mouth is stained white, and then yellow or brown, and the enamel of the teeth is attacked. The soreness and swelling of the mouth and throat; the difficulty of swallowing and of respiration; the thirst and salivation; and the excessive pain and distress which are more especially referred, by the sufferer from this poison, to a diffused abdominal pain rather than at the pit of the stomach or epigastrium, as in the case of sulphuric acid poisoning,—are the most prominent symptoms. The air tubes are also more likely to be affected by the volatile fumes of nitric acid rather than by the nonvolatile sulphuric acid, which does not produce this effect unless by direct contact with the liquid. Sulphuric acid is also more liable to produce constipation, and nitric acid to cause diarrhea. The eructation of nitrous vapors from the stomach is a characteristic symptom. The vomitus is yellow rather than brown or black, as is the case in sulphuric acid poisoning. The abdominal pain may be entirely absent, as in two recorded fatal cases. If the person survive long enough, large portions of the lining membrane of the fauces and œsophagus become detached and are thrown up, together with altered blood and shreds of mucus. Similar matters may be discharged by stool. The diluted acid occasions the same symptoms in a more moderate degree.

101. Subacute symptoms.— As in the case of sulphuric acid, stricture of the air passage and of the gullet arising from secondary inflammation may occur. Although the immediate corrosive effects of the acid may be recovered from, death may occur subsequently from exhaustion and the injury done to the digestive powers. Dr. Black referred, in some remarks before the Royal Medical and Chirurgical Society of London, to the case of a girl, who, "in a fit of despondency, swallowed some strong nitric acid. She left the hospital, but died many months afterwards, but so altered in appearance, that she resembled a woman eighty years of age. She was kept alive for seven weeks entirely by spring water." The œsophagus was nearly closed by the strictures which had resulted from the healing of the ulcerations produced by the acid.[22] The period at which this poison proves fatal varies, therefore, according to its dilution, from a few hours to several months.

We should not expect any of the mineral acids would be used in attempts of murder of adults, on account of the difficulty of administering so repulsive a substance, the resistance to which would offer great obstacles. Nitric acid is used so largely in the arts, and in the manufacture of celluloid, dynamite, gun cotton, etc., and by dyers and hatters in their factories, and, also, in the preparation of absorbent cotton, that the danger of accidental injurious effects should not be overlooked. In spite of this presumption Witthaus and Becker record in their book on Medical Jurisprudence 54 cases of death by poisoning by this acid, of which 13 were homicidal, 34 suicidal, and 7 accidental. They found 65 cases of poisoning noted in the medical journals, 11 of which recovered.

102. Fatal dose.— Two drams is the smallest quantity known to have destroyed life. This was nearly the quantity swallowed in a case reported by Dr. J. M. Warren. Death ensued on the fourteenth day. (It is stated that 3 drams were taken into the mouth, but most of it was spit out.) Dr. Taylor refers to the case of a boy who died in thirty-six hours after taking 2 drams of this acid. An instance in which it was poured into the ear of a sleeping person, and caused death after some time, is related by Dr. Morrison.[23] Mr. James Haywood, a chemist, lost his life by inhaling the fumes of mixed nitric and sulphuric acids, in consequence of the breaking of a carboy which held the mixture. The symptoms, which did not appear for more than three hours, consisted mainly of difficult respiration. Death

[22] Lancet, 1850.

[23] Dublin Jour. Vol. IX. p. 98.

took place in eleven hours. On examination, a considerable effusion of blood was found in the bronchial tubes, and their lining membrane and that of the trachea were congested. The larynx was not examined.[24]

103. Duration of symptoms.— As in the case of other corrosive poisons, death from nitric acid may occur within a few hours (from two to twenty-four hours) or after several months, according to the quantity and strength of the acid, and the vigor of the patient.

104. Treatment.—It is evident, on account of the rapid corrosive action of the strong acids upon human tissues, that any attempts to neutralize an acid which has been taken into the mouth must be offered immediately to prevent its further injurious effects. Alkalies largely diluted with water should be swallowed,—without the use of the stomach tube,—and the patient should be encouraged to vomit up the contents of the stomach; after the neutralization of the acid, demulcent and oleaginous drinks should be freely swallowed. During convalescence from subacute and chronic poisoning, it may be necessary to employ rectal injections of nutriment to aid in the general nutrition, on account of the serious interruption to digestion from destruction or injury to the mucous membrane which lines the primæ viæ.

105. Post-mortem appearances.—These are similar in effect to those which have been mentioned in the case of the preceding acids, except in the color or stains of the eschars and erosions; these are, when the stronger acid has been in contact with the tissues, orange-yellow; and the sloughs and ulcerations are not so deep nor hard, but are pultaceous and are accompanied with shreds of loose mucous membrane. Nitric acid shows, on post-mortem examination, somewhat the same primary lesions which we marked as following the ingestion of sulphuric acid; but nitric acid corrosions cause an orange-yellow or yellow stain on the tissue with which the pure concentrated acid comes in contact, while the dilute acid stains these tissues a grey-yellow or black shade; as these consist of a sphacelation of the lining membrane, they are easily detached, and the subjacent surface is found even and glistening.

If the person has survived several days, they may have been already cast off. The same appearance is found in the fauces, and more or less of it in the œsophagus. The stomach is softened, sometimes perforated, its internal surface is of a greenish-yellow, but sometimes of a black, color, owing to the erosion of vessels and the

[24] Lancet, April 1854, p. 429.

effusion of blood, and the mucous membrane is ulcerated or destroyed. In Dr. Warren's case, the patient was a negress, who swallowed the poison with the hope of destroying her child, supposing that she was pregnant. She died on the fourteenth day. On dissection, there was observed great rigidity; upon the middle of the tongue, a large, yellowish, smooth patch; some redness of the epiglottis. The œsophagus was healthy for the first two inches, but below this it was found exceedingly soft, of a greenish color internally, purple externally, and full of coagulated blood. The stomach was in a similar though much worse state; externally, it had the same purple color, and was universally adherent to the neighboring parts by recent lymph, except at the left extremity, where there were old and close adhesions to the spleen; internally, it was of a greenish-yellow color, emphysematous, and so perfectly softened and pliable that it could not be separated from the surrounding parts without giving way in every direction; the anterior face became detached from the rest of the organ to a great extent when the abdominal parietes were raised; its cavity was filled with recent coagulated blood, and the open orifices of several vessels were distinctly seen on the inner surface. The intestine contained blood throughout the first 2 or 3 feet, but was otherwise uninjured. In a case observed at the Hôtel-Dieu, at Lyons, the stomach was distended with gas, and perforated in the cul-de-sac; the opening, however, was partially plugged by the spleen, which had become adherent over it, but which itself was much corroded. In the small intestine, there were numerous sloughs. In another case, where the person survived the poisoning fifty-four days, the stomach was converted into a vast abscess, with fungous walls made by adhesions among the adjacent viscera. The natural shape and structure of the organ could no longer be distinguished.

In a case of poisoning by nitrous fumes,[25] the endocardium was found much inflamed; the lungs were nearly destroyed, so that the lung tissue was in many places soft like jelly. One portion of the left lung showed marked congestion and was stained yellow. The blood in the lung tissue had an acid reaction and the odor of nitrous fumes. Free nitric acid was detected in the lungs, but not in the other organs.

106. Chemical examination.— Nitric acid may be readily known by its physical properties already mentioned, and by the red fumes which are given off when it is poured upon copper, tin, or mercury

[25] Ann. d'Hygiène, 1875, p. 345 (Vol. XI. 4th ed. p. 81, Nitric Acid.)

in contact with air. Fortunately, nitric acid, unlike sulphuric and hydrochloric acids, is not a normal constituent of the animal economy, so that its detection in toxicological cases is much easier than that of the other two acids, which are normally present in the animal fluids and tissues in the form of sulphates or chlorids, although there are no reagents which precipitate it. Hence it is not so important to distinguish between the free and the combined acid.

Nitric acid, when free, can easily be detected by the following tests: (1) A piece of copper introduced into the acid will be dissolved, forming a greenish-blue solution of nitrate of copper, and red fumes will be evolved. If the solution be very dilute, warming will facilitate the reaction. If the nitric acid be combined with a base, as nitrate of potassium for example, it can be freed from its combination by warming with sulphuric acid. (2) A crystal of ferrous sulphate forms a brown compound with nitric acid. If the nitric acid is free, the brown compound will form around the crystal. If it be combined, the best way to peform the test is to dissolve a small crystal of the sulphate of iron in the fluid and then pour concentrated sulphuric acid down the side of the vessel, so that it forms a separate layer of fluid at the bottom; the nitrate is decomposed at the junction of the sulphuric acid and fluid, and a brown zone forms at this point. (3) A solution of sulphate of anilin gives a purple or violet color, when treated in the same way as ferrous sulphate with nitric acid or a nitrate. (4) A crystal of brucin or a solution of a salt of brucin gives an orange color with nitric acid. (5) Metallic gold is insoluble in pure nitric acid, but is readily soluble in a mixture of nitric and hydrochloric acids (aqua regia). Therefore, if to a solution of nitric acid or to a nitrate, a little hydrochloric acid be added, and a piece of gold foil be introduced into the mixture, it will be dissolved, and we will obtain a solution of trichlorid of gold, which can be tested by the appropriate tests.

When organic mixtures, like vomitus or contents of stomach, are to be tested for nitric acid, the yellow stains, intensely acid reaction, and nitrous fumes and odor will usually leave no doubt as to its presence. In order to isolate it, however, the fluid to be tested should be treated with an excess of calcium carbonate (chalk), which forms with the nitric acid nitrate of calcium, which is a deliquescent compound and readily soluble in alcohol. This mixture should then be evaporated to dryness upon a water bath, the residue extracted with strong alcohol and filtered; evaporate the filtrate upon a water bath, when a residue of impure nitrate of calcium will remain; this can be dis-

solved in water and tested by the above tests. This solution can also be decomposed by carbonate of potassium or carbonate of sodium, and the characteristic crystals of the nitrate of potassium or nitrate of sodium obtained upon careful evaporation of the filtrate. These crystals should always be taken to court as the corpora delicti in any criminal case.

107. Stains on cloth.— The spots made upon colored cloth by nitric acid are more or less yellow, but become brown after a time, the texture of the cloth is destroyed, and the spot, unlike that made by sulphuric acid, soon becomes dry. To determine the presence of nitric acid, the stain may be cut out and digested in distilled water. If the liquid have an acid reaction, it should be then neutralized with potash, and allowed to crystallize. The crystals may be examined as before, by heating with copper turnings and moistening with sulphuric acid. An unstained portion of the cloth should be examined in the same manner. Stains made by nitric acid will not furnish evidence of its presence after a few weeks have elapsed, the acid being much less permanent than the sulphuric. Hence the necessity of proceeding at as early a period as possible to the examination of any suspicious stains upon a dress. Dr. Christison, however, has obtained evidence of the presence of the acid in stains on cloth made seven weeks before, and Dr. O'Shaughnessy after an interval of some months.[26] *

IV. Nitrohydrochloric acid.

108. In general.— Nitrohydrochloric acid (U. S. P.) consists of 180 parts of nitric acid and 820 parts of hydrochloric acid. This is called by the names "nitromuriatic acid" and "aqua regia." It is a golden-yellow-colored, fuming, and very corrosive liquid, having a strong odor of chlorin.

109. Preparations.— Diluted nitrohydrochloric acid should not be made by diluting the strong acid compound, but by adding to 40 parts of nitric acid, 180 parts of hydrochloric acid and 780 parts of distilled water.

110. Action.—Both the stronger and the diluted compound acid have free chlorin, and, in consequence, the ingestion of either would have, besides the action of the two acids of which it is composed, the added danger of the action of this corrosive gas upon the passages to the larynx and throat.

Its action, when concentrated, will coagulate albumin, turn it yel-

* Guy's For. Med.

low, and dissolve it; it will also destroy all vegetable and animal matter, with the evolution of reddish nitrous vapor. The dilute preparation only is used as a medicine in doses of 5 to 8 drops (30 to 50 cg.).

111. **Antidotes.**— A general antidote to all the mineral acids, only as an agent to neutralize the acid, would be an alkaline carbonate, preferably magnesia; syrup of lime or green soap might also be used.

V. Hydrofluoric acid.

112. **In general.**— Fluorin is a normal constituent of many animal tissues, and exists in some to a considerable extent, so that its mere detection is not proof positive of any medico-legal importance. For organic materials free from silica, the best test is to dry the material, fuse with a little sodium carbonate and nitrate, and then heat the residue gently with sulphuric acid in a platinum crucible, covered with a watch glass partly coated with wax or paraffin. The glass will be etched if fluorin, now in the form of hydrofluoric acid, was originally present. If silicon is present at the same time, Taunmann's method is preferable: The substance is mixed with quartz sand, and placed in a flask provided with a three-hole stopper, sulphuric acid is added, and a stream of dry air carries any silicon fluorid into a narrow vessel containing water, where the fluorid is decomposed into gelatinous silicic acid.

We can find but one case of fatal poisoning by this acid, reported by Dr. King. This was a case of a drunkard forty-six years of age, who used hydrofluoric acid to etch on glass. He swallowed about two teaspoonfuls, and died in great agony within thirty-five minutes; he made strong efforts to vomit, had cold sweats, a small pulse, thin and rapid, contracted pupils, and was unable to swallow. After the pulsation at the wrist had become imperceptible and the heart's action had ceased, he continued to breathe.

113. **Post-mortem appearances.**— The autopsy of this one case revealed a large quantity of thick and blackened substance which resembled treacle; when this was removed from the stomach, and the latter was washed out, a reticulation of the lining membrane was seen; the summits of the rugæ were raised and the depressions between them were injected; punctate ecchymoses were marked. The mucous membrane of the mouth was white and softened, with the epithelium partially detached and hanging in shreds.

VI. Boric acid.

114. In general.—A white crystalline body, with a slightly bitter tatse, odorless, soluble in water, and used as a preservative (see Appendix, § 880).

This chemical and antiseptic agent has been in use commercially, and has the reputation of being an injurious drug to man. The active discussion by Chittenden,[27] Liebreich,[28] British Commission,[29] Tunnicliffe,[30] and others, on the one hand, would seem to leave this drug, as a poisonous agent out of a work on toxicology; while the experiments of Annette,[31] Foster and Schlenker,[32] Doane and Price,[33] which seem to show disturbances of digestion, do not appear to indicate that these results end fatally. The Agricultural Department at Washington is investigating the subject of its injurious action by experiments on the human being with food products into which this agent is introduced.[34]

Dr. Luff[35] mentions serious and fatal action following its use as an antiseptic wash, in its injection into pleural sac, abscess cavity, and even into the stomach. These symptoms are general depression, rash on body, vomiting, diarrhea, and, finally, collapse; and, also in nonfatal cases an urticarial or erythematous eruption has generally been seen.

115. Tests.— After fusion with sodium carbonate and nitrate, boric acid forms borates. The fused mass can be extracted with alcohol and sulphuric acid. The alcohol is then ignited, and burns with a green flame which shows four lines in the spectrum.

[27] Am. Jour. of Physiology, 1898.

[28] Vierteljahrsschrift für gericht. Med. 1890.

[29] *Ibid.*

[30] Am. Med. March 13, 1902.

[31] Lancet Nov. 11, 1899.

[32] Report of Kober on Milk Preservatives, U. S. Senate Commission 1902.

[33] Bulletin No. 86, Maryland Agricultural Experimental Station, Sept. 1902.

[34] Since writing the text of this deleterious agent, the editors have received from the Bureau of Chemistry of the United States Department of Agriculture, through the courtesy of the Secretary of that Department and the Chief of the Bureau, a copy of Circular No. 15, which is a digest of Bulletin No. 84, and presents certain conclusions of the effects of boric acid and borax on digestion and health.

The bulletin itself, which is not yet published, is the first report of an investigation of "the character of proposed food preservatives and coloring matters, to determine their relation to digestion and health, and to establish principles which should guide their use."

In the light of probable medico legal bearing of suits at law, in reference to the injurious effects of food preservatives on health of persons purchasing the same in ignorance of the substances contained in foods, the summary of the results of experiments, as given in the circular above named, is published in the Appendix to this volume, p. 797, for the information of medical experts, as well as others of the medical profession interested in this research. We are informed that complete details of the investigation contained in Bulletin No. 84 may not appear in print for some time.

[35] Op. cit. Vol. I. p. 146.

Borates with hydrochloric acid stain turmeric paper red-brown, and caustic potash changes this color to blue-black.

Solutions of $CaCl_2$, Fe_2Cl_6, $AgNO_3$, $BaCl_2$, etc., give precipitates, as a rule white, when added to solutions of borates.

The detection of boric acid or borates in organic matter is rather unsatisfactory, as they are more or less volatilized in extraction. The best method is careful evaporation and drying of the material, preferably in vacuo, and then fusing it with sodium carbonate and nitrate.

VII. Alum.

116. In general.— Alum is largely used as a mordant in calico printing, also in dyeing, and in other arts. Formerly, the most common compound met with was potash alum, the double sulphate of potassium and aluminium, but recently the ammonium compound has replaced the potash one, on account of its lesser cost. Its use in whitening bread, made from poorer quality flour, is condemned by practical hygienists; but its use in baking powders can hardly be considered harmful, as it is completely decomposed if mixed with a sufficient quantity of sodium bicarbonate, in the raising and baking of bread.

117. Properties.—Potassium alum, $K_2Al_2(SO_4)_4+24H_2O$, forms regular transparent octahedral crystals, soluble in water. On heating to redness, it loses nearly half its water and is known as burnt alum. The potassium is easily replaced by sodium, ammonium, etc., forming various alums.

118. Dried or calcined alum.— There are reported a few cases of poisoning by alum, about five only, one in England, four in France. The action is that of a local irritant and corrosive, frothing at the mouth, vomiting, and depression (Taylor). As alum generally provokes immediate vomiting, when given in large amounts, it is not unfrequently combined with some other irritant emetic, and prescribed by physicians who desire to promptly evacuate the contents of the stomach. Though it is not ordinarily considered by medical practitioners to have a poisonous character, yet it is considered as a poison by Mr. Taylor in his treatise, and even Orfila admitted that in animals a large dose operated fatally, destroying life in a few hours. We might make the general statement that, if a considerable dose of alum, say more than an ounce, is swallowed and retained in the stomach, it will act as a strong irritant, and produce serious constitutional disturbance corresponding to the extent of this irritation. It is, on the

authority of Orfila, undoubtedly absorbed into the blood of animals, and may be recovered from the urine.[36] Tardieu, however, does not mention this drug in his treatise on Poisons. Higuet relates a case of poisoning by alum,[37] and at the autopsy found the lesions of an irriant poison.[38-39]

In all these cases the alum was mistaken for salts, sulphate of magnesium.

119. Symptoms.— The following case[40] which occurred May 15, 1872, will illustrate the class of symptoms to be met after poisoning by alum. The symptoms of the above case, in which 30 grains dissolved in a glass of water were swallowed by V. M. at about midnight, were a feeling of constriction and burning in the mouth, throat, and stomach; nausea, followed with one attack of bloody vomiting; no movements of the bowels; extreme discomfort followed, then unbearable anguish; pulse was small and frequent; respiration quick; repeated attacks of syncope; the mind and senses were unaffected. Attributing all these symptoms to an unusual attack following the ingestion of Epsom salts, magnesium sulphate, the patient did not send for a physician until morning; he arrived at seven o'clock and found the patient in a most desperate condition; pulse intermittent and wiry; skin cold; deglutition almost impossible. Death occurred an hour after the arrival of the physician.

Peterson and Haines quote a case, reported by Kramolik, of a young man, aged thirty years, who, by mistake, drank a mouthful of a 10 per cent solution of alum. Neither the mouth nor the throat showed any marked reaction to the irritant, but the patient vomited thirty-nine times within the forty-eight hours following. Palpation of the stomach was painful. Mucus was found in the vomitus, mingled with blood, imparting a chocolate color to the mass. The urine was stained by blood, and showed numerous red blood cells, a few white blood cells, and a few hyaline casts. Traces of albumin were present. Patient was ill for at least thirteen days.

120. Treatment.— There have been so few cases of actual poisoning from alum that no precise treatment for its effects has been made by writers on toxicology. It is suggested by the writer that, as its primary action is that of a caustic, the rules for treatment should be the

[36] Ann. d'Hygiène, 1845, 2 p. 433.

[37] Bull. gén. de Thér. Aug. 1873, p. 137.

[38] See, also, an account of the same case in L'Union Médicale, No. 64. 1873, and Ann. de la Société méd. et chir. de Liège.

[39] See a small treatise, "The Injurious Effects of Alum upon the human system when used in bread or baking powders, and as proved by experiments on man and animals." London. 1879, 8vo. 36 pp.

[40] Ann. d'Hygiène Publique, 39, p. 192.

same as when an alkali caustic has been swallowed; that is, after the natural vomiting has removed the substance swallowed, the stomach should be washed out with large quantities of water; afterwards demulcent drinks, milk, etc., should be administered.

121. Action.—One of the effects of the alum salts, after the taking of which death has occurred, seems to be that of an irritant caustic. In addition, Paul Siem[41] has made a careful research for the absorbed poisonous substances, and without the consideration of the local effect upon the primæ viæ. These researches were concerned with animals, and the substance used was sodium and aluminium lactate, a double salt, equal to 15.2 per cent of alum. He injected 2 to 3 centigrams subcutaneously to frogs, who died in from ten to twenty-four hours afterwards. The symptoms of poisoning were restlessness and, finally, general paralysis of the nerve centers; the circulation was not affected, the heart being the last to die.

He experimented on warm-blooded animals by subcutaneous injections of the double tartrate of sodium and aluminium, after a single dose thus administered to cats of 0.15 gram per kilo weight. These animals died, with symptoms commencing ten to twelve hours after the injection. His experiments would seem to show that the lethal dose for rabbits was 0.3 gram per kilo of body weight; for dogs 0.25, and for cats 0.25 to 0.28 gram. After medium doses, the appearance of the symptoms was delayed for three or four days, which consisted of inappetence, constipation, emaciation, languor, and a disinclination to move. Vomiting and loss of sensation of pain followed, power of swallowing even saliva was lost, and a condition supervened similar to bulbar paralysis.

122. Lethal dose.— There have been too few cases of poisoning by ammonia alum to distinctly lay down the size of a lethal dose. If we follow the experiments above related on animals, in whom, by the way, this poison was administered subcutaneously and was probably all absorbed, an average-sized man would require, to be fatally poisoned, a dose of about 3 ounces, which should be absorbed in the system in one dose to produce its fatal effects. We, of course, are excluding the local caustic or irritant effects on the digestive tract.

123. Post-mortem appearances.— There is but one case in which a careful autopsy is reported:[42]

Decomposition slight; rigor mortis absent. Bluish discoloration of cutaneous veins of the thighs. The scalp, skull, and sinuses were anemic; meningeal veins contained a little blood; brain substance

[41] Schmidt's Jahrbuch, Vol. CCXI. 128. [42] *Ibid.*

normal. Mucous membrane of the mouth, pharynx, and œsophagus covered with a grayish-yellow coating, and easily detached; tongue and tonsils swollen. An effusion of coagulated blood was observed between the trachea and œsophagus, and in front of the thyroid cartilage. Peritoneum inflamed throughout; the cavity contained considerable brownish serous fluid. Omentum injected; its veins and those of the mesentery full of black blood, and hard. External surface of stomach grayish and strongly injected; veins filled with black blood, which was hard and friable, as though due to the action of acids or astringents. Mucous membrane gray and shriveled; at the pyloric end disorganized and inflamed. The contents of the stomach consisted of a brownish fluid which attacked the polished surface of the scalpel; near the pylorus was an abundance of gray powder. The organ was somewhat contracted. Duodenum thickened, contracted, and of a gray color; in its upper part was more of the gray powder. Several bright-red exudations were observed under the peritoneal covering of the small intestine; notable injection of both the large and small intestines. In the first half of the latter were two oval gangrenous spots measuring 10 and 15 centimeters in the long diameter. No well-marked alteration in the large intestine; the descending colon contained some semi-solid fecal matter of a greenish-brown color. Liver small, grayish, and hard; its peritoneal coat covered with a fibro-albuminous exudation of a yellowish-gray color; its substance anemic. Biliary ducts contained calculi as large as a peppercorn. Gall bladder dilated, hypertrophied, and gray; it contained some pale-yellow bile, in which swam about 130 calculi as large as peppercorns. Spleen small and contracted. Kidneys much injected. Bladder empty. Lungs normal; pleural cavities contained considerable bloody serum. Heart enlarged; walls thin; cavities contained some soft clots like currant jelly.

Chemical examination of the stomach and intestine proved the poisonous substance to be potash alum, which had probably been calcined.

124. Baking powder.—Mr. Blyth, who, with most English writers on toxicology, considers that the danger of being poisoned from the use of alum mixed with baking powders is not likely, sums up this subject in the following words: "Alum baking powders, containing from 30 to 40 per cent alum mixed with bicarbonate of soda, are in commerce, and have been for a long time, many tons being sold yearly. When water is added to such powders, decomposition takes place, result being sodic sulphate and aluminic hydrate, carbonic acid being

given off." It may be pointed out, also, that, with regard to the possible lethal effect of a noncorrosive salt of alum, presuming that the lethal dose for man is the same as that for a cat, the amount of aluminium to kill a 68-kilogram man would have to be equal to 17 grams.

125. Tests and detection.— Soluble aluminium salts give, on the addition of sodium or potassium hydrate, a bulky, white, flocculent precipitate, which is soluble in excess of the reagent; with ammonium hydrate, a similar, white precipitate, which is almost insoluble in excess of the reagent,—especially in the presence of ammoniacal salts.

Sodium phosphate gives a bulky precipitate of aluminium phosphate, which is soluble in an excess of sodium or potassium hydrate, but almost insoluble in ammonium hydrate,—especially in the presence of ammonium chlorid. This precipitate is soluble in mineral acids, but not in acetic acid.

For the separation of alum from organic matter, the material can be ignited in a platinum dish, the ash extracted with hydrochloric acid, excess of acid evaporated, a few drops of nitric acid added, and then the solution filtered. From the filtrate the aluminium is precipitated as a hydrate by the addition of ammonium hydrate, and boiling. The precipitate is washed several times by decantation, finally on a filter; transferred to a platinum crucible, ignited, and weighed as Al_2O_3. To confirm the mass in the crucible as being Al_2O_3, it is fused with $KHSO_4$, allowed to cool, and extracted with boiling water. It should completely dissolve. Any residue is SiO_2, which should be dried, ignited, and weighed, and its weight subtracted from the previous one.

The solution, after the fusion of the Al_2O_3, and extraction with boiling water, can then be tested for aluminium by the above tests.

Further, in testing for alum, where possible, a quantitative estimation of the sulphuric acid should be made, in addition to the tests for aluminium.

VIII. Ammonia.

126. Properties.—Ammonia gas, (NH_3), is colorless, with a strong, irritating, pungent odor, a strong alkaline reaction to moist test paper, and very soluble in water; strong solutions of the gas have the above properties like the gas. It is very irritating to the mucous membrane of the nose and throat and air passages.

Ammonium carbonate is a white, crystalline body, soluble in water,

and is used as a caustic. The solid has the property of giving off fumes of ammonia gas, and is therefore irritating.

127. Preparations.— (U. S. P.) Aqua ammoniæ; aqua ammoniæ fortior; linimentum ammoniæ; spiritus ammoniæ; spiritus ammoniæ aromaticus; glycyrrhizinum ammoniatum; linimentum camphoræ compositum; smelling salts (sal volatile); also contained in much diluted form in ammoniated tincture of guaiacum, and in ammoniated tincture of valerian.

128. Action.— There are a few cases where ammonia vapor has caused serious and fatal symptoms to life. Two cases are mentioned by Mr. Blyth; one, where the injudicious use of ammonia, applied to the nostrils, was administered to an epileptic during stupor; the other, by the breaking of a bottle of ammonia water, and the sudden escape of a large amount of this caustic gas. During the manufacture of gas by the ammonia process, vapor may escape.

This volatile alkali has occasionally been used with homicidal purpose, but, in general, cases of poisoning by it are the result of accident. The vapor of strong ammonia has destroyed life, when respired too long a time by a person in a state of temporary insensibility. The effects produced by swallowing a strong solution of ammonia are somewhat similar to, though more intense than, those of the other alkalies. Plenck relates that a man, who had been bitten by a mad dog, had administered to him a glassful of ammonia. His lips, tongue, and gums turned black immediately, and he died within four minutes.[43] In another case, strong ammonia was taken with suicidal intent. The symptoms were collapse, serous and bloody diarrhea, bloody vomiting, excruciating pain in the abdomen, and death in six hours. The mind remained clear till the last.[44] Two cases are reported in the same paragraph of children who were killed by accidentally swallowing a mixture of hartshorn and sweet oil.[45] A little boy, two years of age, took about half an ounce of very pungent spirits of hartshorn from a bottle. He immediately screamed and was very sick, bringing up, at first, stringy mucus of a light color, and then some more which was dark. The lips were swollen, the breathing was harsh, hurried, and somewhat obstructed, and afterwards became somewhat croupy. There was no insensibility nor any diarrhea. He recovered.[46] In another case, reported in the same journal, 1 ounce

[43] Mitscherlich, Lehrbuch, II. 277.
[44] Ann. de Thérapeut. III. 443.
[45] Times and Gazette June, 1855, p. 353.
[46] Iliff, Lancet, 1850, Vol. I. 337, Am. ed.

was taken in milk, by a man who supposed it to be castor oil, having poured it out in the dark. He took immediately copious draughts of warm water, and vomited a quantity of matter like soapsuds. The inside of the mouth, the upper lip, tongue, and fauces were white, and other parts excoriated; there was great difficulty in swallowing. He said he felt as if he were on fire from his stomach to his mouth; his voice was husky, pulse small and frequent, and the surface cold. He was ordered dilute acetic acid and demulcent remedies, under which he soon recovered. There was no diarrhea throughout the case.[47] A woman took a wineglassful of strong liquor of ammonia by mistake for the acetate, yet in a fortnight she was convalescent.[48] Dr. W. Reed has reported the case of a man who swallowed, by mistake, 3 drams of the strong solution of ammonia, and an equal amount of the sesquicarbonate dissolved in 2 ounces of oil. The symptoms resembled those above described; but they subsided, and were relieved under appropriate treatment in about eight days.[49]

Chevallier relates an instance of an attempt to poison with ammonia. The mistress of an officer, he being desirous of breaking up the connection, at their last proposed interview attempted to make him swallow a quantity of ammonia. A physician was sent for immediately. He found the lips excoriated, with phlyctenæ, the tongue swollen and deprived of its epithelium, and the mouth and palate abraded. The throat was so sore as to prevent swallowing, and pressure on this and the region of the œsophagus was very painful.[50]

As may be gathered from these cases, acute poisoning by ammonia is marked by the local caustic effects of a corrosive, and these local effects may be so violent as to cause death by shock. The following additional case, reported by McGillam,[51] is offered as a further illustration of acute poisoning by ammonia water. A man swallowed half a pint of liquor ammoniæ, and was brought into the Great Northern Hospital four and a half hours later. Mustard and oil were first given to the patient, and afterwards followed by acidulated mucilage. There was great difficulty in swallowing. Ten hours from the time of taking the ammonia, the patient was hoarse and in a drowsy condition, with a dusky countenance; the skin was cold, the body temperature being 100.8°, pulse rate 130; the patient had freely vomited mucus and blood. Twenty hours after the ammonia had been swallowed, the lips were much swollen, tongue very dry, and mucous mem-

[47] *Ibid.* 1852, I. p. 374.

[48] Wilkins, Lancet, 1846, I. 385.

[49] Times and Gazette, July, 1855, p. 59.

[50] Am. Jour. of Med. Sci. April, 1854. from Gaz. des Hôpitaux.

[51] Med. Times and Gazette, London, Dec. 1878.

brane of the mouth in parts eroded; the fauces was red and congested and so swollen that there was hardly any open space between its two sides, the mucous membrane of the throat being edematous, softened, and inflamed. There was great pain in the abdomen, and tenderness on pressure, with marked tympanites. The patient had several stools during the previous night, but passed no blood, though he twice had vomited blood; he complained of burning pain in throat and at pit of the stomach. After thirty hours had passed, the patient became comatose, and had great difficulty in breathing, the respiratory movements being 66, and the pulse 180 to the minute; diarrhea had been present all day. He died in forty-eight hours after taking the dose.

Another case of fatal poisoning occurred in France, and was communicated in detail by M. Albert Robin to the Société de Biologie.[51a] The patient, a woman, drank 45 grams, about an ounce and a half, of a solution of ammonia (strength not stated). Prof. Gubler remarked that, in this case, besides the usual symptoms which are above described, she had an abundant flow of saliva, and albumin in the urine; she also suffered from quite severe muscular pains, especially in the thigh. Her death occurred after forty-five hours of suffering.

The deaths from ammonia, as shown by the table of Mr. Blyth, given on page 15, first chapter of this work, amounted to 98 during ten years, in England and Wales; of these 64 were by accident or negligence, and 34 by suicide. But the deaths occurring in Massachusetts, as shown in our table in the same chapter, were 4 only, of which 2 were by accident or negligence, and 2 from suicide.

The solution of ammonia and water, varying in strength, and sometimes named liquid ammonia or spirits of hartshorn, causing, on swallowing, a strong, burning sensation in the mouth and gullet, which is succeeded by a sense of heat and pain in the stomach, vomiting of a watery liquid containing particles of mucus stained with blood, shows a caustic effect on the local tissues with which the alkali comes in contact. Signs of the inflammatory action upon the alimentary tract are noticed by violent attacks of diarrhea, accompanied with tenesmus at the anus. The secondary irritant action on the mucous surface can be recognized by the edematous swelling and redness of mouth, throat, and the entrance to the air-passage; this is marked by symptoms of distress and noisy, asthmatic breathing, and the efforts, by coughing, to remove the particles of mucus detached from the swollen mucous membrane. If the caustic solution is

51a Feb. 14. 1874.

strongly concentrated, the shock to the system from intense local irritation, produces a collapse, which is accompanied with a cold, clammy skin, and marked by a thin and feeble arterial pulse. In the case of liquid ammonia, the respiratory passages may be so strongly attacked by free ammonia gas not completely held in solution, because passing from a comparatively cold condition to a higher temperature produced by the heat of the mouth and throat, that the intense, local action upon its mucous surface may cause adhesions, and, by preventing the entrance of atmospheric air, produce asphyxia. It is quite evident that attempts to remove the irritant poison from the stomach by the use of a stomach pump through the primæ viæ will seriously injure the inflamed mucous surface, and is especially to be deprecated. The efforts for the purpose of neutralizing the alkaline action of the caustic should be limited to administration of a dilute solution of vinegar or of lemon juice, followed by bland liquids such as flaxseed tea and olive oil.

Two fatal cases of death, caused by drinking ammonia water accidentally swallowed by mistake, are reported by medical examiners in Massachusetts in the year 1898. A waiter brought a strong solution of ammonia to a customer who asked for an "ammonia cocktail." An invalid, who was in the habit of taking digitalis in aromatic spirits of ammonia, and of inhaling the vapor of strong ammonia, mistook the latter for the former bottle, and drank from it; she died from asphyxia, because the edematous swelling of the air passages prevented the inhalation of atmospheric air into the lungs. The strong caustic action on the mucous membrane in the mouth made it appear, at the autopsy, as if this surface had been scraped with a comb.

Washing fluid often contains a solution of strong ammonia, also caustic potash or soda. Medical Examiner Harris reports (case 27, 1893) the death of a child eighteen months old, from drinking a strong solution of ammonia used as a washing fluid. The immediate cause of death was edema of the larynx.

129. Subacute poisoning.— Should death not occur within one or two days after taking the poison, we would have a set of secondary symptoms as a result of subacute poisoning by ammonia. These symptoms may not all of them be found in one case; for instance. Dr. Francais[52] reports a case of suicide by ammonia. The details of his case are similar to those above mentioned. He concludes that ammonia acts not only as a local irritant and rubefacient, but also

[52] Bull. de Société de médecine légale de France, Vol. V. 1873, p. 32.

causes secondary corrosive action by imbibition or absorption into tissues in the vicinity of the alimentary canal, and deep-seated gangrene which may invade the spleen, liver, and kidneys. There was no corrosive action upon the lips or mouth. His patient lived seven days.

It should also be remarked that, after the ingestion of weak solutions of the alkali, in which case the intense local effects may be very mild, the absorption of alkali, especially ammonia, into the blood, may be followed by signs of anemia which may be serious enough to require treatment by appropriate medicines to counteract this tendency. The writer has in mind a case in which a very prolonged and persistent treatment of this character was required: A child of the age of eleven had swallowed, by accident, quite an amount of ammonia water, the local effects of which were counteracted by the acidulated and mollifying drinks above indicated, so that only a slight local inflammatory condition remained, and in a few days, cleared up. Persistent anemia remained, which was treated by iron preparations. These could not be well borne, at first, in ordinary doses; but by beginning with very small doses of the weaker preparations, and gradually increasing the size of the dose, the patient gradually recovered her general health, the anemia being relieved. It should also be observed that, even if the patient survive the effects of the local irritant and caustic action upon the inlets of the body, the secondary inflammations may, as in the case of the acid corrosives, obstruct the air passages or gullet; and that, moreover, like arsenic, phosphorus, carbolic acid, and other poisons, fatty degeneration may occur in the tissues of the vital organs, thus inducing a fatal result from impairment of their functions. This fatty degeneration may occur as early as the second twenty-four hours, and the rapidity of death will depend upon the extent of this degeneration.

130. Post-mortem appearances.— Ammonia irritates the respiratory organs, and edematous condition of the glottis may occur. Schmans states that "a croupous exudation may be found in the air passages and on the œsophagus; in other respects it causes lesions like other alkali." These are rather soft, white or gray eschars, which are not readily detachable from the underlying deeper tissues; they are not brittle like the eschars produced by the strong acids, and are not so often followed by secondary ulceration. Schmans states that "when large quantities remain in the stomach until death and after, the eschars and the intact parts between swell, and the stomach appears very soft and semi-transparent. The affected portions gradually be-

come a greasy mass of a brown or black color. The stomach may be perforated; similar conditions may be found in the intestines, and adjacent viscera are cloudy and then transparent." The mucous membrane of the mouth, throat, œsophagus, and stomach is softened, inflamed, and generally eroded in patches; from the fact that the vessels are partially destroyed, blood, red or dark-colored, may stain these patches.

The post-mortem appearances which are found in the intestinal canal are more extensive than those which are observed in the case of the caustic acids, and the corrosions have not the same blackened or charred character, but are soft and moist; in acute poisoning these moist eschars and the diffused softening extend over the whole mucous surface. When death has been delayed, ulcerous or purulent inflammations may be observed, the partial healing of which may cause the contractions or constrictions of the gullet above referred to; in this case also, the neighboring tissues may be softened, unless the repair has been completely effected.

131. Chemical examination.— In cases of poisoning by ammonia or its compounds, the chemical analysis must be performed very soon after death, on account of the liability to error from the formation of ammonium compounds by the putrefaction of the tissues. It can be recognized, when present in the form of ammonium hydrate (ammonia water) or ammonium carbonate (1) by its odor, (2) by white fumes which are evolved when a glass rod moistened with hydrochloric acid is brought near the suspected fluid, and (3) by the alkaline reaction of the vapors emanating from the fluid, as shown by bringing a piece of moistened red litmus or turmeric paper near the surface of the fluid. (4) A drop of platinic chlorid upon a watch glass moistened with a little hydrochloric acid and exposed to the vapor will soon contain yellow crystals of ammonio-platinic chlorid, which, when examined under the microscope, are seen to be dodecahedral crystals, like those of the corresponding potassium compound. This test can also be applied to a liquid as detailed under potassium (see § 140). (5) Tartaric acid gives the same result as with potassium compounds, except that, as in the case of the above test, it can be applied to the vapor as well as the liquid. If antidotes have been given, or if the ammonia has been neutralized in any other way, we can free it from its combinations by warming the compound with a little milk of lime, and test the vapors as above mentioned.

Ammonia is separated from organic mixtures by distilling the mixture in a retort, after rendering it alkaline with sodic hydrate.

and, preferably, adding alcohol to the mixture in the retort; this facilitates the volatilization of the ammonia vapor, and also prevents the destruction of the organic matter (and consequent formation of ammonia) by the sodium hydrate. The distillate can then (after testing for ammonia by the reaction, odor, and fumes with hydrochloric acid) be neutralized, and the amount of ammonia in it be estimated, if desirable.

IX. Miscellaneous alkaline caustics.

132. Properties of potassium hydrate and sodium hydrate.—Potassium hydrate, KOH, is a grayish-white, amorphous substance, freely soluble in water and alcohol, but not in ether; its solution has a soapy feeling and a strong, alkaline reaction. It deliquesces rapidly, and absorbs carbon dioxid from the air, forming potassium carbonate.

Sodium hydrate, NaOH, is similar to potassium hydrate in most of its properties. The sodium carbonate, however, is not deliquescent like potassium carbonate, but is efflorescent. This sodium carbonate, commonly known as "sal soda" or "washing soda," is a crystalline body, freely soluble in water, with a strong, alkaline reaction.

133. Action of potash, soda, and their carbonates.—These two alkalies and their carbonates may be mentioned under one head, since their poisonous effects are similar. Eau de Javelle (hypochlorite of sodium, or chlorinated soda, colored red with peroxid of manganese) is mentioned by Tardieu,[52a] as having caused death by suicide and by mistake. This solution is used largely in laundries for bleaching purposes, and, consequently, exposes many temptations to suicide or careless mistake from the readiness with which it may be handled. One hundred and fifty or two hundred grams (5 to 7 fluidounces) will produce fatal results. Caustic potash or soda (liquid) in the dose of ½ ounce (two to five teaspoonfuls) is also fatal. Potassa dissolves in half its weight of water, soda in a little less than twice its weight; hence a solution of caustic potassa and soda may contain a large amount of the caustic alkalies. Our notice of them will be brief, as they seldom occasion poisoning, and, when they do, it is almost necessarily accidental, and its cause is readily known. They may prove fatal either by their immediate action or by their remote influence upon the system. When swallowed in large quantity and in a concentrated solution, the taste is exceedingly nauseous and acrid, and a sensation of burning heat and constriction in the throat, œsoph-

[52a] Sur l'Empoisonnement, 1875, p. 274.

agus, and stomach is excited. When a considerable portion of the solution has reached the stomach, there follow great pain and tenderness in the abdomen, vomiting of mucus and altered blood, inability to swallow, copious diarrhea, and general collapse. If the patient survives a few days, the inside of the mouth is seen to be greatly inflamed, sloughs become detached from the throat, vomiting continues, there is a dysenteric condition of the bowels, and the patient dies from marasmus. When the dose is too small to induce an immediate fatal result from the acute irritant, and corrosive action of the caustic, the patient will experience a very painful gastric catarrh, or active inflammation of the mucous membane, resulting oftentimes in a constriction of the gullet, which may interfere with deglutition, and thus cause death by starvation.

Life may be, however, prolonged even for months and years, and the person finally die from the impairment of his digestive powers.

A case is related by Dr. Barham, in which a caustic solution of impure carbonate of sodium (soap lees) was swallowed by mistake, and the patient died two years and three months afterwards in consequence of stricture of the œsophagus caused by it.[53] Several instances of a similar kind are recorded, and Sir Charles Bell relates one in which death did not take place until twenty years after the accident. A more recent example is one furnished by Dr. Deutsch.[54] The quantity drunk was a "portion of a glass," and was estimated to contain ½ ounce of caustic potassa. The early symptoms were such as those described above, but partial recovery took place. Gradually, however, swallowing became difficult, and death took place in twenty-eight weeks after the accident. The lower part of the œsophagus was found enormously thickened, so that its canal was nearly obliterated near the stomach. The increased thickness was entirely confined to the mucous membrane, the muscular coat, on the other hand, having nearly disappeared.

Washing fluids often contain a solution of caustic potash or soda, as well as ammonia. Medical Examiner Harris (Massachusetts, case 183, 1900) reports the death of a child nineteen months old, who chewed a rag used for washing windows, which was soaked with caustic potash. It died within twenty-four hours. No autopsy.

134. Action of potassium carbonate and soap lees.— Potassium carbonate, or salt of tartar, has a similar action to that of caustic potash, though it is not so powerful in its action. Soap lees, used in a prepa-

[53] Lancet, 1850, Vol. II

[54] Times and Gazette, May, 1858, p. 537.

ration of soft soap, contains caustic potash and consequently, produces the same effect.

135. Symptoms of alkali poisoning.— At first, after swallowing, a burning sensation is felt in the mouth and gullet; second, heat and pain are experienced in the throat, gullet, and stomach. Vomiting usually occurs, of a bloody fluid with shreds of mucous membrane. Diarrhea with tenesmus usually appears in the majority of cases. The lips, tongue, and throat are swollen and red. The patient is distressed with difficult breathing, and with the attempt of getting rid of swollen and detached portions of the mucous membrane. Cold, clammy surface, and collapse, with a small and feeble pulse, end the scene.

136. Diagnosis of alkali poison.— It is difficult from the symptoms to determine whether the corrosive action of the poison swallowed is due to an acid or alkali. The best means to determine this is to test the secretions of the mouth or the vomited material by means of litmus paper, or by a differential diagnosis. It will have been noticed from the description of poisoning from the mineral acids that their action upon vegetable matter is quite easily seen by their behavior in attacking soft and vegetable matter, which is unlike that of an alkali.

137. Lethal dose.— "The smallest recorded fatal dose of caustic potash is 40 grains." (Luff.) The shortest time is which caustic potash has resulted fatally is three hours.

138. Treatment.— We have already mentioned the danger of using the stomach pump or tube, where the caustic action upon the mucous substances of the mouth and throat has softened that tissue. When we know that we are dealing with a caustic action of an alkali, attempts should be made to neutralize the alkali by a weak acid, such, for instance, as vinegar diluted with water, lemon juice, or a weak solution of citric or tartaric acid. Stimulants should be administered by subcutaneous injection of ether, brandy, or morphin. After neutralization of the free alkali, emollient drinks of oil, milk, or flaxseed tea should be freely administered.

139. Post-mortem appearances.— These have already been described under the head of the action of ammonia, since the action of the caustic alkalies is very similar.

140. Chemical examination.— This depends upon the detection of large amounts of the alkalies in the vomitus, contents of stomach, or other fluid examined; and, since salts of the alkalies, such as common salt, cream of tartar, etc., are such common articles of food and

medicine, and many are also normal constituents of the animal economy, it is, of course, necessary to isolate them in their original form, and separate them from their chlorids or other neutral salts. First, the reaction of the fluids to be examined should be ascertained; if any of the above substances are present, it will be strongly alkaline. The amount of alkali present can be determined by exactly neutralizing a portion of it, after extracting with distilled water, with a solution of some acid (preferably oxalic or sulphuric) of known strength. If the poison consists of one of the alkaline hydrates, it can be separated from most of the salts by extracting with alcohol. (Potassium carbonate is also soluble in alcohol, but sodium carbonate is insoluble.) In any case of poisoning by an alkali, the substances to be submitted to chemical examination should at once be placed in a tightly stoppered bottle, so that no air can gain access to them, on account of the liability of the alkaline hydrates to absorb carbonic acid from the air and become converted into carbonates. If potassium hydrate or carbonate were the poison, it may be detected by the following tests: (1) By the alkaline reaction. (2) By exactly neutralizing a portion with tartaric acid, and then adding as much more tartaric acid as was required to neutralize it in the first place. This will precipitate the potassium in the form of a white crystalline compound (cream of tartar). The reaction is facilitated by shaking, or by adding a little alcohol. (3) Compounds of potassium impart a violet color to the colorless gas flame. (4) A drop of the concentrated solution acidulated with hydrochloric acid and treated with a drop of platinic chlorid, will yield a yellow crystalline precipitate, which may be seen under the microscope to consist of dodecahedral crystals. Ammonium compounds yield similar crystals, but they can be expelled by previous evaporation and ignition. This platinum compound should always be produced as the corpus delicti in cases of poisoning by potassium compounds.

Sodium hydrate and carbonate are chiefly recognized by the strongly alkaline reaction, the intense yellow color imparted to the colorless flame,—this flame giving the characteristic spectrum, which is different from the potassium spectrum,—and by the absence of any potassium or ammonium compounds, both of which can be precipitated, while sodium forms soluble compounds only with the ordinary reagents, such as platinic chlorid and tartaric acid.

X. Antimony.

141. Preparations.—(U. S. P.) Antimony oxid, antimony sul-

phid, purified antimony sulphid, antimony peroxid, antimony trisulphid, purified antimony trisulphide, compound antimony pills, sulphurated antimony, wine of antimony, compound syrup of squill, antimoniated powder.

Witthaus and Becker state that out of 119 cases of fatal poisoning 5 per cent were suicide, 14.3 per cent were homicides, and the remainder were caused by negligence or accident.

Compounds of antimony give rise to very few cases of poisoning compared with arsenic. The only compounds of antimony which have any toxicological importance are the tartrate of antimony and potassium (tartar emetic), and the trichlorid of antimony. Metallic antimony has been thought to be poisonous, since it has caused injurious symptoms when inhaled in the form of vapor; but, as Christison suggests, these symptoms may have been due to arsenic with which antimony is almost always contaminated.

Tartar emetic, which is so largely used in medicine, has given rise to most of the cases of antimony poisoning. It is a white crystalline solid readily soluble in water. It may cause both acute and chronic poisoning, some of the cases of criminal poisoning recorded being of the latter variety. Of the preparations used in medicine there are the tartar emetic itself, the wine of antimony, which contains 4 parts of tartar emetic in 1000 of the wine (U. S. P. 1880), and the compound syrup of squill, which contains 3 parts of tartar emetic in 2000 parts of the syrup (U. S. P., 1880).

142. Symptoms of acute poisoning.— The salt is capable of producing violent and alarming symptoms, and occasionally, also, fatal effects. Its immediate action upon the stomach appears to be irritant, since it produces a burning pain in the stomach, excessive vomiting, and diarrhea. The large doses which have been tolerated in some febrile affections, such as pneumonia, rheumatism, and mania à potu, have thrown some doubt upon its irritant properties, and its speedy rejection from the stomach in other cases defeats, in a measure, both its local and constitutional poisonous effects.

The phenomena of acute poisoning by this agent may be thus described: The patient is attacked, within fifteen minutes or half an hour after ingestion, with incessant retching, precordial cramps, and burning heat, distention of the epigastrium, watery and frequent stools, dryness of the throat, difficult deglutition, an unpleasant metallic taste in the mouth, and sometimes a copious discharge of saliva. The mucous membrane of the mouth becomes coated with a white aphthous crust like that of canker, which finally becomes brown or

black in color; when this condition is seen in the mouth, we may also expect to find it, if the case ends fatally, in the stomach and intestines. Soon after, the patient complains of distress at the pit of the stomach, and becomes faint, or falls into a state of syncope, of agitation, of vertigo, or of a sort of drunkenness. The region of the stomach continues painful, and there is heat in the throat and a difficulty in swallowing; vomiting reappears with extreme facility, and the vomited matters are sometimes mixed with blood; the urine is scanty; the extremities become cold, and the skin is covered with a clammy perspiration; the depression is very great, and often there is loss of muscular power; agitation continues, and sleep is broken; the pulse, moreover, is a little quickened, but regular and soft. About the fourth or fifth day there appears upon the extremities and on different parts of the body a vesiculo-pustular eruption like that caused by antimonial ointment. In aggravated cases, the vomitings give place to a persistent hiccough, the stools become involuntary, the urine is suppressed, the coldness of the extremities extends and becomes general, the extremities become cyanotic, delirium and convulsions ensue, and death approaches in a period of time varying from three to six days. In children, death may occur much earlier, sometimes after a few hours.

In a few rare cases the ingestion of an excessive dose of tartar emetic does not cause vomiting, but may be followed by alvine evacuations, complete prostration, collapse, a few convulsive movements, and death in a few hours.

Most often a single excessive dose does not cause death in a person poisoned by this agent. The abundance and suddenness of the vomitings oppose the absorption of the poison, and the reaction, which may occur very early, limits the symptoms of poisoning to their first period; after having vomited for several hours, accompanied by alvine evacuations, the symptoms abate, the tongue becomes red, the pulse full, hard, and frequent, and the surface of the body warm. There remains for a short time a headache, epigastric distress, and a feeling of lassitude. The patient recovers his usual health in ten days or a fortnight.

Peterson and Haines state that in exceptional cases, when no vomiting occurs for an hour and where drowsiness and loss of muscular power come on early, among other symptoms mentioned above, tetanic spasms may occur, and, later on, persistent inflammation of the lower bowels and loss of hair. Trichlorid of antimony also causes corrosion, on account of its strongly acid reaction; this corrosion occurs on the mucous membrane of the digestive tract.

With a knowledge of symptoms following continued, fractional, small doses of antimony, persons have attempted suicide or murder. Symptoms of chronic poisoning resemble those of natural diseases, such as loss of voice, weak pulse, albuminuria, catarrhal inflammation, prostration, and disturbances of the digestive organs. In the case of Mrs. Pritchard, who was poisoned by her husband, a physician, by the administration of small quantities of this poison in continuing doses, vomiting was the principal symptom after partaking of food, accompanied with constant thirst, chronic diarrhea, extreme depression, violent muscular cramps; these symptoms improved when she left home, but grew worse on her return. Her husband maintained that she was sick with typhoid fever; she lived for four and a half months in this condition, and finally died, her husband confessing the murder after his condemnation in court.

143. Lethal dose.— According to Taylor, ¾ of a grain (0.048 gm.) is the smallest dose producing fatal results in a child. A single dose of 10 grains (0.648 gm.) is a dangerous dose, and may even be fatal; if the same amount should be given, frequently repeated in fractional doses, it would be more likely to prove fatal. The fatality of the dose is increased, where little or no vomiting takes place; because, under such conditions, the absorption of the poison is limited to what remains in the stomach and is absorbed by the intestines; active diarrhea would also tend to remove large portions of the drug without absorption. This will explain the reports of the many cases where 3 drams or more have been taken in a single dose (11.6 gm.) without causing fatal results. By careful watching by the physician of the depression, the action of heart and circulation may be continued by repeated, pretty large doses, under which the emetic action of the drug is somewhat diminished by an acquired tolerance of the patient.

Death may occur from a poisonous dose within a period of time from six to forty hours. In a few exceptional cases where death has appeared in a shorter period than above, it may have been coincident with the character of the disease during which the drug was administered, and this fact should be borne in mind.

144. Treatment.—Where the drug itself produces free emesis, thus removing from the stomach a large portion of the poison, no other emetic may be given. Where there is not emesis, an active emetic, such as sulphate of zinc, mustard, and the subcutaneous administration of apomorphin may assist in removing this poison from the stomach. Tannin or strong tea, as a drink, will neutralize the antimony by producing the insoluble tannate of antimony, which can be

washed out of the stomach by the use of the stomach tube; hot water should be used in this case. To overcome the depressing action upon the general system due to the absorption of the poison, alcoholic stimulants, and surface application of heat over the regions of the stomach and to the extremities, would tend to increase the circulation of the blood through the small or capillary vessels. The subcutaneous injection of morphin will relieve the pain and distress following the absorption of this drug, enlarge the lumen of the capillary blood vessels, and control the resulting diarrhea from the irritating action upon the lower bowels.

If the form of antimony swallowed is the acid chlorid, the corrosion upon the mucous membrane would contraindicate the use of the stomach tube.

145. Action.— The action of antimony upon the mucous membrane is similar to that of the irritating action of arsenic, and as with arsenic, pustular eruption is produced on the mucous membrane of the whole digestive tract. Its action upon the heart retards the pulsation and reduces the blood pressure in the blood vessels, and, on death taking place, the heart action ceases in diastole,—relaxation of its muscular walls. There seems to be no especial action on the nerve centers; for, if the brain and spinal cord of a frog be destroyed, and the same be poisoned by antimony, the effect on the heart and blood circulation is the same as when the brain and nerve centers are intact. Hermann,[55] in commenting upon certain experiments by Giannussi and Grimm, who attempted to explain the vomiting as due to an inhibition of the nervous centers, is strongly of the impression that the vomiting should be referred to a reflex action due to the inflammatory excitement of the digestive apparatus,—especially of the stomach. This, it seems, whether the poison is administered subcutaneously or injected into the brain. Hermann noted that, when it is introduced into the veins, much larger doses are required to excite vomiting than when it is introduced into the stomach. He explains, therefore, the action upon the digestive tract as being caused by the absorbed antimony being excreted into the intestinal tracts, and, during its passage, exciting the vomiting and diarrhea. He contends that Majendie's well-known experiment,—demonstrating that, after extirpation of the stomach, vomiting movements were noted,—is not opposed to this view.

146. Diagnosis.— Differential diagnosis between the symptoms of

[55] Lehrbuch der experimentellen Toxicologie.

poisoning by antimony as distinguished from that of arsenic is not easy. It may be generally said that the intoxication symptoms of antimonial poisoning are more profound than those from arsenic.

147. Post-mortem appearances.—These are not very dissimilar to those which appear after death from arsenic. The similar appearances are parenchymatous gastro-adenitis; inflammatory changes in mouth (rather more frequent than with arsenic); mucous membrane swollen, and on it may be observed pustules and aphthæ; fatty degeneration of kidneys, liver, heart, and blood vessels. The unlike symptoms,—generally without blood in the stomach, signs of pneumonia observed, absence of degeneration in the muscular tissues.

Taylor refers to certain cases of fatal poisoning seen by Mr. Hartley, in which two children, of the ages respectively of five and three years, swallowed each 10 grains of tartar emetic, the following appearances were noted: The bodies were examined between four and five days after death. "In that of the boy, there was effusion of serum in the right pleura; the lower lobe of the right lung, posteriorly, was redder than natural, and the peritoneum was injected from recent inflammation. The mucous membrane of the duodenum was inflamed and covered with a whitish-yellow viscid secretion; this was observed throughout the intestinal canal, although the color was deeper in the colon and rectum; there was no ulceration. The peritoneal coat of the stomach was congested. The mucous membrane of this organ was much inflamed,—especially about the larger curvature and at the cardiac orifice; there was no ulceration. The contents (about two ounces and a half of a dark grumous fluid, having a slightly acid reaction) were very adherent to it; and in one place there was a patch of lymph. The tests used did not indicate the presence of antimony. With regard to other appearances, the tongue was covered with a white fur and appeared sodden; the fauces were not inflamed; the trachea and œsophagus had a natural appearance. On opening the cranium, the dura mater was found very vascular; the longitudinal sinus contained a coagulum of lymph, and but very little blood. The vessels of the surface of the brain were very much injected with dark blood, the whole surface having a deep purple color. Every portion of the brain, when cut, presented many bloody points. The cerebellum and medulla oblongata were also extremely vascular; there was no effusion in the ventricles or at the base of the brain. In the body of the girl, the morbid appearances were similar; there were also patches resembling the eruption of scarlatina on the arms, legs, and neck. The arachnoid membrane was more opaque than usual;

and on the mucous membrane of the stomach, where the inflammation was greatest, were two or three white spots, each about the size of a split pea, which appeared to be the commencement of ulceration."[56] The body of a woman, who died in seven hours after taking an unknown quantity of tartar emetic, and was examined thirty-nine hours afterwards, presented no lesions whatever in the stomach or elsewhere.[57] But, when life is protracted after the ingestion of the poison, the changes will usually be found like those in the two cases above described, in other words, an aphthous condition of the mouth, fauces, and œsophagus; softening, inflammation, or ulceration of the stomach, and to some extent, also, of the intestines; and generally a dark color and a liquid state of the blood.

M. G. Felizet, in an interesting article,[58] insists that the principal point of interest in the trial of Dr. Pritchard for poisoning his wife[59] from a scientific point of view, is that of furnishing an example of poisoning by tartar emetic, without the appearance of anatomical lesion. Drs. Douglas Maclagan and Littlejohn reported that the body of Mrs. Pritchard presented no appearance of morbid action capable of accounting for death.

Most ordinarily, and especially when a single excessive dose has been taken, tartar emetic produces extended and multiform lesions. The œsophagus appears red and presents some ulcerated spots. The stomach and intestines are the seat of a violent inflammation, characterized by a vivid redness with softening of the mucous membrane, upon which are distributed blackened or brownish-red patches formed by the infiltration of blood and by hypertrophy of the follicles. The internal surface of the stomach and small intestine is covered with a blackish, thick, and viscid secretion, sometimes streaked with blood. Occasionally may be found in the primæ viæ, and even in the commencement of the small intestine, true pustules filled with pus. This inflammation of the intestines is especially liable to be found located in the very upper and lowest portions, the duodenum, cæcum, and rectum.

Antimony produces fatty degeneration[60] of the liver. This fact is so well known in the Duchy of Brunswick that the peasants of that

[56] Taylor, from Lancet, April 25, 1846, p. 460. See, also, a case by Mr. Beal, Lancet, Jan. 21, 1854.

[57] C. Ellis, Boston Jour. Dec. 1856, p. 400.

[58] De l'Action toxique du Tartre Stibie, procès du docteur Pritchard. Archives Gén. de Méd. Sept. 1865.

[59] *Vide* also Boston Med. and Surg. Jour. Aug. 10, 1865.

[60] De la steatose, Thèse du concours d'aggrégation de la Faculté de Médecine de Paris. Blachez, 1866, p. 38.

district feed their geese upon white oxid of antimony to make them fat and well flavored.

This fatty degeneration may also extend to other organs, especially the kidneys and heart, and also to the muscular tissue generally, as in arsenic poisoning. The liver may become so fatty that it resembles the condition known as acute yellow atrophy of the liver, so that some authorities are, at the present time, inclined to believe that all reported cases of acute yellow atrophy of the liver were really cases of phosphorus, arsenic, or antimony poisoning. The writer met with a case a few years ago, which was diagnosticated at the Boston City Hospital from the autopsy as a case of acute yellow atrophy of the liver; analysis of this organ showed the presence of antimony in it, although no tartar emetic or other antimonial compound had been given while the patient was in the hospital; no previous history of his case could be obtained. The pathological action of this poison upon the lungs is in no way different from that of other poisons of the inorganic group, and, consequently, not much importance should be attached to the pulmonary lesions occurring in a suspected case of poisoning by this agent.

Attention has been already called[61] in this work to the liability of confusing the symptoms of poisoning by tartar emetic with those of a disease called cerebro-spinal meningitis; and in the trial of Mrs. Wharton for poisoning General Ketchum, it has been shown that this is a difficult question to decide. It must, however, be suggested that some of the medical men, summoned as experts in that case, may not have stood the usual test of professional qualifications, or may have been prejudiced either from a spirit of rivalry, or by a premature conviction that the prisoner was guilty of the charge of poisoning. The testimony of some of the witnesses was to the effect that the symptoms preceding the death of General Ketchum could not be attributed to any known disease, and yet they seemed to resemble those of the above-named malady, as has been clearly exposed in an article by Dr. Reese, in the American Journal of Medical Sciences, before referred to.

148. Chronic poisoning.— The poisonous effects of tartar emetic, when used in small doses for a long time, have been carefully studied by Mayerhoffer[62] and by Dr. Taylor. The latter toxicologist considers it probable that the secret poisons, which, from time to time, have produced so many victims, and have rendered so many names

[61] *Vide.* Paragraph 31 (g), p. 57.

[62] Beiträge zur Heilkunde, II. 372.

infamous in history, were antimonial. The following description of chronic poisoning by tartar emetic is furnished by Dr. Taylor:[63]

If tartarized antimony be given in small but increasing doses for a long period, there is uneasiness, nausea, and retching, followed by vomiting; the stools are pasty, and diarrhea, attended with thin bilious and mucous discharges, gradually sets in; the abdomen at the same time is distended and tense. The voidance of the urine becomes frequent and violent. The region of the stomach is tender and painful; that of the liver appears fuller, and is sensitive to the touch. There are griping pains in the bowels, with stiffness and pains in the lower limbs. The warmth of the skin is at first increased; there is itching or irritation with alternation of heat and cold. The appetite is suppressed, and when any substance is eaten, there is nausea with an immediate disposition to vomit. A roughness or rawness is perceived in the throat, with painful swallowing; the tongue is covered with a dirty mucus, and the mouth is clammy. The head feels full and heavy. At a still later period, the feces contain much mucus, and are frequently strongly colored with bile. The blood gradually loses its fibrin, and becomes almost liquid, and dark-colored. It contains an increased proportion of fluid and saline matters with traces of antimony. If the use of the substance be discontinued, the action of the heart and pulse becomes slow, with a loss of power; the breathing is difficult, the complexion dusky; there is complete depression of the vital powers with great debility and emaciation; the legs become heavy and stiff, as if paralyzed, and death may follow as a result of the noxious impression produced on the more important organs of the body. Small doses act more powerfully when dissolved than when administered in the form of powder. In addition to the above symptoms, the vomiting, from which the patient suffers, is either attended or followed by the distention of the abdomen, and flatulence; by liquid bilious motions, with colicky pains, paleness, and sunken appearance of the countenance; and by cold perspiration, giddiness, great prostration of strength, incapacity on the part of the patient to raise himself to the erect position, disposition to rest and sleep, loss of strength, fullness and frequency of the pulse, faintings, in many cases a feeling of coldness, accompanied with a heat or flushing of some parts of the body, *e. g.*, the face. If there is a recovery from this condition, pain in the stomach is felt for a long time afterwards, and inflammation of the stomach to a greater

[63] Guy's Hospital Reports, 3d ser. III. 388.

or less degree is set up. After the vomiting, there remains, for a longer or shorter period, an unwillingness to take food, and nausea in partaking of it. Among other effects the perspiration and the urinary secretion are observed to be greatly increased.

If the use of tartarized antimony be continued in increasing doses, when the poison has been already carried into the blood, the secretory organs are more strongly stimulated, and absorption, as well as secretion,—especially of the serous liquids,—is greatly augmented.

If vomiting does not take place after large doses, the following symptoms of poisoning are observed: Metallic taste, nausea, retching, and bilious vomiting; burning pain in the throat, gullet, and stomach; spasms of the jaw and neck; pain and flatulent distention of the abdomen, with frequent watery motions; coldness, pallor, and clamminess of the skin, sometimes great heat of skin; difficult breathing, painful sobbing, giddiness, stupefaction, loss of consciousness, delirium, spasms of the arms and legs, with complete prostration of strength. Death appears to result from the impression produced on the nerves of motion, as well as on the nerves of the lungs and heart, leading either to asphyxia or paralysis.

149. Chemical examination.— It is proper to bear in mind, as Dr. Taylor remarks, that antimony given in a large dose, or repeatedly in small doses, is rapidly absorbed and eliminated chiefly by the urine. It is, at the same time, deposited in a greater or less quantity in the tissues and organs. Under recent administration, if in sufficient quantity, it may be found in the stomach and bowels, and little or none may be present in the liver. After a variable time it disappears from the stomach and bowels, although it may be present in the feces, while the liver, kidneys, and spleen may contain it in large, and the other organs in small, quantity. In certain diseased states of the system, the complete elimination of the metal may require a period of twenty-five or thirty days or longer; but in a healthy subject, to whom only ordinary medicinal doses have been given, the antimony is quickly expelled.

The double tartrate of antimony and potassium (tartar emetic) is readily soluble in water, even when it contains cream of tartar. It is insoluble in alcohol. It crystallizes in the form of small, white crystals belonging to the rhombic system.

If a little of the crystalline powder be heated upon a knife blade or porcelain plate, it blackens, and there is evolved an odor resembling that of burnt sugar, due to the tartaric acid. A black residue having an alkaline reaction to test paper is left.

If a drop of a solution of sulphureted hydrogen or sulphid of ammonium be placed upon a few of the crystals, they will assume an orange color, owing to the formation of trisulphid of antimony. A solution of tartar emetic, if strongly acidulated with hydrochloric acid and subjected to the action of sulphureted hydrogen, will be precipitated, the precipitate having an orange color, and consisting of trisulphid of antimony, which is readily soluble in sulphid of ammonium, or in a solution of potassium or sodium hydrate; it is insoluble in a solution of sesquicarbonate of ammonium, and can, by means of this reagent, be separated from the trisulphid of arsenic; it is soluble in hot, concentrated hydrochloric acid, which does not dissolve the sulphid of arsenic. If not first acidulated with hydrochloric acid, the above reagents will only produce an orange-colored solution.

Exposed to the reducing flame of the blowpipe with carbonate of sodium, white incrustations and globules of antimony are obtained, known by the needle-shaped crystals with which they are beset, and by their metallic brilliancy and brittleness.

Dilute nitric or hydrochloric acid throws down from a solution of tartar emetic a white precipitate, which is soluble in an excess of the reagent and also in tartaric acid, which enables us to distinguish this compound from a similar one produced by the same reagents in solutions of bismuth salts. If just enough acid has been added to redissolve the precipitate first formed, and the resulting solution be thrown into a large amount of water, the white oxychlorid or nitrate will be again precipitated. The same is true of the solution formed by the action of strong hydrochloric acid upon the trisulphid of antimony.

A solution of tannic or gallic acid causes a precipitate in solutions of tartar emetic; hence the value of astringent infusions containing either of these acids, as an antidote in cases of tartar emetic poisoning.

Ferrocyanid of potassium causes no precipitate, as it does in solutions of salts of most of the metals.

150. Galvanic test.— If a solution of tartar emetic be acidulated with hydrochloric acid, and a piece of platinum foil, around which is bent a piece of metallic zinc, be introduced into the fluid, galvanic action will take place, hydrogen gas will be evolved, and the antimonial compound will be decomposed, metallic antimony being deposited upon the platinum in the form of a black sooty coating, which can be dissolved in tartaric or hydrochloric acids, and subjected to the other tests for antimony.

151. Reinsch's test.— This is performed in the same way as for

arsenic. The coating formed upon the copper has, as a rule, a more sooty appearance than with arsenic; and when the copper is heated in a tube closed at one end, the white sublimate is formed much nearer the flame than the arsenical sublimate, and it is usually in the form of prismatic crystals, although rarely it may assume the form of octahedral crystals, as mentioned above in speaking of arsenic. If the copper containing the coating of antimony be boiled with a solution of potassium hydrate, the antimony will be dissolved, and can be tested by adding a solution of sulphureted hydrogen, filtering from the sulphid of copper thus formed, and acidulating with hydrochloric acid, when the orange sulphid of antimony will be precipitated.

152. Marsh's test.— This is performed in the same way as the test for arsenic. The antimonial mirrors obtained, both in the tube and upon the porcelain plate, are usually blacker and less brilliant than those of arsenic.

The following table[64] may be convenient to determine at a glance the different properties of arsenic and antimony:—

Arsenic.	Antimony.
Volatile, easily displaced in a current of hydrogen.	Less volatile, melting into minute globules.
Disappears in presence of nitric acid. The solution, evaporated to dryness, deposits, by a neutral solution of nitrate of silver, a brick-red precipitate of arseniate of silver.	Gives no reaction with nitrate of silver, when treated in the same way.
Is dissolved by the addition of an alkaline hypochlorite.	
The metallic ring, heated in a current of sulphureted hydrogen, gives a yellow sulphid of arsenic, soluble in ammonia, unaltered by hydrochloric acid gas.	Preserves its metallic lustre, if the layer is rather thick. The ring gives an orange sulphid, which is transformed into a volatile chlorid by hydrochloric acid gas.
The gas set free in Marsh's apparatus reduces the nitrate of silver, and gives a soluble, arsenious acid, metallic silver being precipitated.	The gas reduces nitrate of silver, and the antimony is wholly precipitated with the silver.
Is not dissolved by a solution of ammonium sulphid.	Is dissolved by ammonium sulphid.

By this test arsenic can be separated from antimony, when compounds of the two metals occur together, as may be the case either from the combined administration of the two drugs, or from the presence of arsenic as an impurity in tartar emetic which has been administered. M. Bouis states that the best method for separating these two

[64] Médecine légale, par Briand et Claude, Paris, 1869, p. 654.

poisons is the following, based on the action of arseniureted hydrogen, and of antimoniureted hydrogen upon a solution of nitrate of silver:—

Instead of forming a metallic ring in Marsh's apparatus, the gas is passed into a test tube containing a solution of nitrate of silver; the arseniureted hydrogen is transformed into arsenious acid; the antimoniureted hydrogen gives the insoluble antimonide of silver; the two gases produce at the same time reduced silver. When all the suspected matter has been introduced into the apparatus, and the gas has been disengaged long enough, the black precipitate of silver and the antimonide of silver are collected on a filter and thoroughly washed. In the filtered liquid the excess of silver is precipitated by hydrochloric acid in the form of white chlorid of silver; which is again filtered, and the arsenic is precipitated from the clear solution by sulphureted hydrogen. As for the black residue which remains upon the filter, that can be digested by aqua regia, and then diluted with a little water; chlorid of silver is deposited, and in the filtered liquid the chlorid of antimony is left. It would be better, instead of treating the residue with aqua regia, to boil it with a concentrated solution of tartaric acid, which will dissolve the antimony without attacking the silver.

153. Detection of antimony in organic liquids.— If an examination of the tissues is required for the purpose of detecting absorbed antimony, the liver should be selected as the organ most likely to contain it.

The method employed for the destruction of the organic matters is the same as for arsenic, the sulphureted hydrogen precipitating the antimony in the form of the orange-colored trisulphid of antimony; this color is, however, always disguised by the presence of organic sulphur compounds, so that no inference whatever can be drawn from the color of the precipitate produced by the sulphureted hydrogen. This precipitate should be purified in the same way as described under arsenic, by dissolving it in a hot mixture of ammonia water and ammonium sulphid, evaporating to dryness, oxidizing the residue with either concentrated nitric acid, or hydrochloric acid and chlorate of potassium, and evaporating to dryness again. This residue can be dissolved in water to which a little hydrochloric acid has been added, and subjected to all of the tests mentioned above. The spleen, kidneys, and urine should likewise be examined for antimony.

It is evident that the presence of antimony, either in the stomach or absorbed in the other organs, may be due to the proper medicinal ad-

ministration of the salt. The forms under which it is given, and the occasions on which it is prescribed, are numerous, and it is not unfrequently administered in cases of poisoning with other substances, without a thought of the complications it may, in case of a death, place in the way of the chemist. Hence, unless the possibility of its introduction into the system under any of these circumstances be fully excluded, the object of the medico-legal inquiry may be entirely frustrated.

XI. Arsenic.

154. In general.—Arsenic and its compounds is one of the most, if not the most, important poison that we meet with, as it is so commonly used, and access to it is so free.

155. Occurrences.—Among the most common household articles in which arsenic has been found are the so-called "fly poisons," "moth exterminators," and numerous vermin killers. "Rough on Rats" is composed of white arsenic and powdered charcoal. Many kinds of enameled cooking utensils formerly contained arsenic, but, at present, the better kinds are generally arsenic-free. It is often used by those who have to do with horses, in the form of so-called "balls," to improve the coat and appearance of the horse. Soft coal, burned in open fires, may cause symptoms of arsenical poisoning. Arsenic has been found in nearly every article of apparel in common use, such as dress goods, linings, stockings, shoes, slippers, underwear, artificial flowers for hat trimmings, etc. It used to be found in draperies, window shades, and furniture coverings of all kinds, but of recent years the presence of arsenic in such articles has rather decreased. Pasteboard boxes covered with glazed papers, playing cards, glazed paper wrappers for lozenges and candies, and many playthings covered with paper, or painted, often contain arsenic. In articles of food, arsenic has been found in beer.

Arsenic is often used by taxidermists in the form of a powder or soap, and gases arising from the skins of animals preserved in this way may cause arsenical poisoning. Sheep washers and wool washers often use an arsenical soap or "sheep-dip" to kill the parasites in wool. In dissecting rooms, cases of arsenical poisoning have occurred from the "injection mass" used to preserve bodies. Anilin dyes formerly contained arsenic in considerable quantities, and, at present, arsenic is often found in them,—especially in the red dyes,—and of course articles colored with such dyes may give rise to symptoms of poisoning.

The following medicinal preparations contain arsenic:—

Liquor potassii arsenitis (Fowler's solution); liquor sodii arsenitis (Harle's solution); liquor sodii arsenatis (Pearson's solution); and liquor arseni et hydrargyri iodidi (Donovan's solution); all these may cause arsenical poisoning.

Most so-called "cancer-paste cures" contain arsenic in large amounts, and many "tonic" pills also contain arsenic as one of their constituents.

Of the pigments used commercially, containing arsenic in combination as a salt, may be mentioned Scheele's green (copper arsenite), which is commonly used in paints, also for coloring glazed papers such as are used on book covers, toys, etc., and also for coloring artificial flowers, window draperies of all sorts, etc.

Paris green (Schweinfurt green) is a mixture of the acetate and arsenite of copper. It is very largely used on potato plants and tobacco. The latter use is dangerous, however, as the leaves are the part of the plant which is consumed.

Orpiment, As_2S_3, is used as a yellow pigment sometimes, and has caused arsenical poisoning.

There has been considerable discussion as to the cause of arsenic poisoning, when the source of the arsenic was an arsenical pigment in wall paper. It is now generally agreed that a volatile arsenical compound is produced by certain molds, such as the mucor mucedo, aspergillum glaucum, and others, in the presence of arsenic, moisture, and a temperature from 60° to 95° F. It is not arseniureted hydrogen, but probably an organic combination of arsenic oxid. Wall paper containing more than a tenth of a grain of arsenic to the square yard is not allowed by the laws of Massachusetts to be sold, and this amount is probably as high as is consistent with safety.

156. Properties.—Elementary arsenic is a steel-gray, crystalline substance with a bright, metallic lustre, and is very brittle. It also exists as a dull-black, amorphous powder. It is generally stated that it sublimes at 180° C. In a dry atmosphere it is stable, moisture oxidizes it slowly, and by heat it is easily converted into the trioxid. It forms addition products with Cl, Br, I, Cu, and Fe. It combines readily with nascent hydrogen. Hot sulphuric and nitric acids are decomposed by arsenic with the formation of arsenious and arsenic oxids. Hot hydrochloric acid with arsenic forms the trichlorid, a more or less volatile liquid.

Arseniureted hydrogen, hydrogen arsenide, arsin, AsH_3, is formed by the action of nascent hydrogen upon arsenic compounds capable

of reduction. It is a colorless, combustible gas with a garlicky odor The flame is bluish white, and, when pressed against a cold surface, deposits elementary arsenic as a brown-black powder. It reduces silver nitrate solutions. It is the most poisonous of the inorganic compounds of arsenic.

Arsenious oxid, As_2O_3, improperly called arsenious acid, is a white, crystalline body, soluble in water, forming arsenious acid, with a slight acid reaction, a sweetish, metallic taste. It sublimes at 218° C. It also occurs, when fused, as a white, transparent, vitreous, amorphous body, somewhat less soluble in water than the crystalline form. Nascent hydrogen converts it into arsin, while oxidizing agents convert it into arsenic oxid. With metallic bases it forms salts called arsenites, while with acid radicles it acts as a base.

Arsenic oxid, As_2O_5, is a white, amorphous, deliquescent body, soluble in water, forming arsenic acid.

Arsenious sulphid, arsenic trisulphid, As_2S_3 king's yellow, orpiment, is a yellow, crystalline body, soluble in alkaline hydrates, ammonium polysulphid, but insoluble in dilute acids.

Arsenic disulphid, As_2S_2; realgar, is a red, crystalline substance. soluble in alkaline sulphids and in hot potassium hydrate.

157. Preparations.—(U. S. P.) Arsenious acid (arsenic trioxid, white arsenic); liquor acidi arsenosi; liquor potassi arsenitis; solution of arsenic and mercuric iodid (Donovan's solution); liquor sodii arsenatis; liquor arseni et hydrargyri iodidi.

Out of the 110 total cases of fatal cases of poisoning by arsenic, as shown in Blyth's table, 2 were for murder, 57 were suicides, 51 from accident or negligence. In the table of the medical examiners' returns for Massachusetts, out of 419 cases of fatal poisoning for the twenty-five years, 10 were homicides, 380 suicides, 29 by accident or negligence; of these 10 cases of murder by arsenic, 5 were by one woman who used this poison to attempt to obtain the benefits of a life insurance policy, which served as the basis of our remarks upon the general attempt to defraud life insurance companies of the value of a life policy. There are several points of interest to the toxicologist, which were brought out in the evidence adduced during this trial, and which are of great importance. Some of them refer to the complication arising from the use of embalming fluids injected after death into the bodies of the victims; some to the fact that death occurred after a commencing convalescence from an attack of pneumonia; some to the filing of certificates of death for another cause than that of poisoning; some to the evidence that the vomitus and

liquids, which appeared upon the clothing of the deceased and upon the bedclothes, were not reserved for examination by the experts called to investigate circumstances connected with the deaths of the various victims; some to the question as to whether the arsenical poison might not have been received by the victims in an accidental way, either from the iron works in which one of them was a workman, or from arsenical wall papers. We invite our readers to examine the details of the published report of this trial.[65-6]

158. General remarks.—The attention of the reader is especially called to the details of fatal poisoning by various forms of arsenic, which are reprinted in the Appendix to this volume, as in the previous edition.

Arsenic is contained in most of the vermin poisons, rat poisons, and fly paper. It seems also to have been the poison selected for several historical cases for poisoning by wholesale, of which the case of Mrs. Robinson of Massachusetts furnishes an example. This is undoubtedly due to the fact that the poison under any of the various substances named above is readily accessible,—especially in the United States,—and because it has no disagreeable taste, and is not easily detected in the food swallowed by the victim. We have especially noted, in our first chapter, its use as an embalming fluid, which is known to criminals, as a further difficulty in the evidence of chemical expert testimony, on account of which it is difficult, in cases of embalming, to determine whether the poisoning was administered before death or placed in the body after death. We would refer our readers to case No. VIII. mentioned in the Appendix, where this important point was brought out in the trial for murder of Mrs. Robinson. Prof. Holland, in Peterson and Haines' work on toxicology which we quote *verbatim:* "The reports of registrar general of England show fifty-one deaths from arsenic in England and Wales for five years. Counting the number of poison cases, thought worthy of record in the medical journals of the world in 1880–89, arsenic figured in 8.3 per cent, poisonous foods in 8.7 per cent, and lead in 10.5 per cent. It is stated by Witthaus that in the decade 1879–89, there were 12 indictments for murder by poison in 31 counties of New York state, in 6 of which the poison alleged to have been given was arsenic, in 1 it was strychnin, and neither morphin nor phosphorus was mentioned.

[65-6] The official report of Sarah Jane Robinson for the murder of Prince Arthur Freeman, in the supreme judicial court of Massachusetts; published by the Attorney General, under chap. 214 of Acts 1886; Wright and Potter Publishing Co. State Printers. 1888, pp. 112–115.

Analyzing 820 cases, he found 45.2 per cent homicides, 27.3 per cent suicides, and 27.5 per cent accidental. In the absence of rigid restrictions upon the sale of arsenical compounds such as are imposed by other governments, the United States has a bad eminence in this respect. The Massachusetts reports give a list of 114 deaths in ten years in that state alone. Only 9 of these were proved to be homicidal. There were 35 suicides from arsenic in New York city in one year (1891)."

The following nonofficial preparations, quack remedies, and other substances containing arsenic, are taken from Mr. Blyth's work on toxicology:—

Arseniate of iron, containing 33.6 per cent of metallic arsenic.

Pilula Asiatica, containing 1/12 grain (5.4 mgm.) of arsenious acid in each pill.

De Valanguis' solutio Solventes Mineralis, containing 0.034 per cent AsO.

Veterinary arsenical medicine:

- A ball for worms containing 1.3 per cent arsenious acid.
- A common tonic ball containing from 5 to 10 per cent (.324 to .684 gm.) arsenious acid.
- An arsenical ball for improving horses' coats, containing 2.5 per cent arsenious acid.
- Linseed meal and treacle containing 8 grains (.518 gm.) of arsenious acid.

Rat and fly poison, etc., arsenical paste for rats, 5 per cent arsenious acid.

Another rat poison containing 46.8 per cent white arsenic.

Fly poisons:

- "Papier moure" containing arsenious acid.
- Dark gray powder containing metallic arsenic which has been exposed some time to the air.
- Other mixtures containing solution of arsenious acid of uncertain strength, sweetened with sugar, treacle, or honey; also, of a mixture of arsenious acid, trisulphid of arsenic, treacle, and honey.

Quack and other nostrums:

A celebrated arsenical paste for external use in cancer.

Arsenious acid	8 per cent.
Cinnabar	70 per cent.
Dragon's blood	22 per cent.

Frère Come's Cancer Paste.

Arsenious acid	1 part.
Charcoal	1 part.
Red mercury sulphid	4 part.
Water	q. s.

Davidson's Cancer Remedy.

Arsenious acid	50 parts.
Powdered hemlock	50 parts.

Pigments, etc.:

King's yellow, trisulphid of arsenic or orpiment, contains from 10 to 20 per cent arsenic acid.

Mineral blue contains equal parts of arsenite of copper and potash.

Emerald green (Schweinfurt green) is a cupric arsenite and acetate.

Scheele's green is a hydrocupric arsenite containing 52.8 per cent of arsenious anhydrid.

Sheep washes: Simply solutions of arsenic mixed with soft soap, potash, and soda ash, etc.

Arsenical soaps; Becour's arsenical soap contains 20.2 per cent arsenic.

Arsenical compounds used in fireworks are contained in the following:

"Blue fires"	5 per cent.
"Green fires"	14 per cent.
"Light green fires"	1.75 per cent.
"White fires"	.76 per cent.

A highly interesting case of homicidal poisoning with it has been reported by Dr. Schütte.[67] The wife of a barber, named Dombrowsky, was suddenly attacked, on the morning of the 11th of April, with violent vomiting and purging, with pain and heat in the epigastrium, and excessive thirst. A physician was called to visit her in the evening, but, being ignorant of the cause of the attack, prescribed simple remedies. She had no fever, her pulse was slow and soft, and the abdomen was not tender upon pressure. The vomiting, and especially the purging, still continued; and although they afterwards abated, her strength sank rapidly, and she died on the sixth day. On the third day after death an examination of the body was made. There was not found any natural cause of death. Evidence of the

[67] Casper's Vierteljahrs. Oct. 1854.

presence of arsenic was obtained by the usual liquid tests. By Marsh's apparatus, arsenic was detected in the fluids contained in the stomach; and the amount obtained from this, and from the subsequent analysis of the stomach itself, was computed at 19 grains. Additional circumstantial evidence, which fixed the crime upon the husband, was derived from the examination of a few particles of a shining, black powder found in the extreme end of his pocket, and also from the analysis of the dried, spilled contents of a cup of sago, which, intending to give to the deceased, he had placed in the stove to warm, but which had been cracked by the heat. It was also proved conclusively that he had himself purchased, at several times, portions of arsenic and of cobalt, which were found in the house. This fact, it may be remarked, was clearly brought home to him by the commendable provision of the Prussian Code, which requires that a person purchasing poison shall give a receipt therefor to the apothecary, containing his name, address, the date, and also the alleged purpose for which it is required. These receipts were produced upon the trial. The prisoner was convicted chiefly upon this and the admirable and minute chemical investigation, and sentenced to be beheaded.

Metallic arsenic, when perfectly pure, is not poisonous, but its poisonous effect in the above cases is explained by the fact that, when exposed to the action of a moist atmosphere, it becomes partially oxidized and changed to the form of white arsenic. This mixture was formerly much used as a fly poison, under the name of fly powder. Metallic arsenic is also partly oxidized when placed in water, the white arsenic which is thus formed is dissolved in the water, and this solution has also been used as a fly poison under the name of fly water. The principal cases of poisoning by metallic arsenic (with the exception of the case mentioned above) have been due to the ingestion of these fly poisons by children.

The poison which is generally known under the name of arsenic, or white arsenic, is one of the oxids of the metal, and has a slight acid reaction. It is met with in one of two forms,—either as a white, vitreous, crystalline substance, or as a white, opaque, and granular powder. It is sparingly soluble in cold water (about 1 in 500 parts), but is more freely dissolved by boiling water, which takes up the acid in about the proportion of 1 of the acid to 10 or 12 of water.[68] Dr. Taylor found, by numerous experiments, that a fluidounce of hot water took up, in cooling from the boiling point, nearly one grain and a quarter of white arsenic—about 1 part in 400 of water; but that, if

[68] Will's analysis.

boiled for an hour on the poison, and allowed to cool, the water held dissolved about 12 grains to the ounce—about 1 part in 40. In some experiments made by Schroff upon the solubility of arsenious acid, he found that 1 part of the acid in 480 of water, the liquid being frequently shaken, was not fully dissolved in fourteen days. He boiled 1 part of arsenic with 100 of water, and found that complete solution took place after fifteen minutes' boiling.[69] It is even less soluble in some liquids containing organic matter. The different statements with regard to its solubility possibly depend upon the difference in this respect between the crystalline and the opaque powder. Its taste is not, as was formerly represented, acid, but, on the contrary, is, when at all perceptible, rather sweetish. It is sometimes described as bitter, usually rough, etc. As a general rule, it may be stated that it is without taste, except when in solution, when the taste perceived may be faint and sweetish. The sparing solubility of this substance is the cause of its want of taste, and also explains the fact that, in the majority of cases of poisoning by it, it is found in larger or smaller quantity in the matters vomited, or adhering to the mucous coat of the stomach. Its solubility is increased by the presence of an alkali or an alkaline carbonate. It is also much more soluble in hydrochloric acid than in water.

This poison gives rise to a very large number of cases of poisoning, criminal and suicidal, as well as accidental. The number of cases met with in Europe has much diminished of late years, on account of the numerous restrictions placed upon its sale and purchase by law; but in this country arsenic is so largely used as a preservative for hides and other substances, for stuffing birds, and in many manufactures, that it is a very easy matter for any one who desires it to obtain possession of a large amount of arsenic without any record being made of the purchase. This ease with which it may be obtained, and its freedom from taste, so that it cannot be detected immediately upon being taken into the mouth, render it a favorite poison for the criminal.

In 1854 Dr. Tschudi published an account of the toxicophagi of the lower countries of Austria and Styria. These persons were in the daily habit of taking large doses of arsenic, with the effect of improving the freshness of their complexion, and becoming more active and free in respiration.[70] When this practice was suddenly discontinued, emaciation and gastric disturbance ensued.[71] The accounts

[69] Canstatt's Jahresbericht für 1853, Bd. V. p. 52.

[70] Wiener medizinische Wochenschrift.

[71] Med. Times and Gazette, London, July, 1854, p. 66.

given by Tschudi and Vogt[72] have been opposed by Pereira, Christison, and others,— especially Taylor,[73] who cites reasons why the fact of arsenicophagism, true or false, can be of no practical value to the medico-legist. Prof. La Rue[74] gives an interesting account of a man who was in the daily habit of eating and smoking arsenic, without producing any but favorable symptoms. In the presence of the professor he swallowed three grains of pure arsenious acid, then a minor dose weighing a grain and a half. Another half grain he smoked, mingled with his tobacco, filling the laboratory with the odor of garlic. The next day he swallowed 4 grains of arsenious acid, and on the day following was perfectly well and free from any gastric disturbance. There can be but small doubt that the arsenic in these cases of arsenic eaters is absorbed; in fact, Dr. Maclagan had some urine of toxicophagi collected and chemically analyzed, the arsenic being by this means recovered.[75] The medico-legal bearings, however, of such instances have a very definite aspect; because the habitual use of any drug—arsenic, opium, morphin, quinine, or any other except hydrate of chloral—causes the consequence of acquiring tolerance by the gradual increase from a small, safe dose to that which, in a person unaccustomed to its use, may be a fatal dose. It results, therefore, to a simple question of evidence whether, in any given case of doubtful poisoning, the victim has by habit become tolerant of what are ordinarily considered fatal doses.

It is pretty commonly known that grooms and horse dealers frequently give arsenic in the corn, oats, or other food to horses; this practice increases the assimilation of food, and makes the horse plump, fat, and with a brilliant coat. They also take a piece of arsenic about the size of a pea, tie it up in a cloth rag with a little oatmeal, and fasten it to a bit; the saliva, dissolving the poison slowly, assists in its absorption. Shot also contains a little arsenic, and about a half pound to a pound of shot is also administered to horses by tying it in a bag to the bit. Horses, accustomed to being dosed with small amounts of arsenic, lose their health and good looks by persistent continuance. It is probable that the arsenic, by stimulating the glands concerned in the digestion of their food, increases their secretions by stimulation of the cells contained in the glands. Arsenic is equally noxious when inhaled in the form of vapor, or applied externally to a denuded surface, or upon the mucous membrane of the

[72] Med. Jahrb. des oster. Statts, 1822, I. 99.

[73] On poisons, London. 1859, p. 91; and Med. Jur. Phila. 1883, 185.

[74] Boston Med. and Surg. Jour. June 28, 1866, p. 439.

[75] Edin. Med. Jour. Sept. 1867.

vagina or rectum.[76-7] Its effects are extremely rapid when it is inhaled, but when it is absorbed from a wounded surface, the symptoms usually do not occur so soon as when it is swallowed. Being an ingredient in most of the pastes used by cancer curers in the extirpation of scirrhous breasts, it is by this means not unfrequently introduced into the system, and has produced death with all the symptoms of arsenical poisoning. Even the small proportion of arsenic which is contained in the stearin of some candles, has, when the latter has been used for the purpose of dressing a blistered surface, produced nausea, pain in the stomach, thirst, redness of the tongue, spasms of the muscles, weakness and irregularity of the pulse, and death within twenty-four hours.

Several cases are quoted by Christison from Fodéré and others, where arsenic, given by injection into the rectum, proved fatal; and, introduced into the vagina, caused death in less than twenty-four hours. It is said, moreover, to have produced violent symptoms when applied to the unbroken skin, as when used as a depilatory.

A case of death from the external application of arsenic to the head of a child two years of age, affected with porrigo favosa, is related by Dr. McCready, of New York. A woman obtained about half an ounce of arsenic, and, mixing it with gin, rubbed it well into the heads of several of her children affected with this disease. It was followed by redness and swelling of the face; in the child alluded to, however, it produced diarrhea and tenesmus, with paralysis of the lower extremities, but no signs of local inflammation. The mother stated that she had, on one previous occasion, applied the arsenic, and, though the application was followed by some swelling, this soon subsided, and the head seemed much better.[78] Dr. Mitchell, of Liverpool, relates a case in which characteristic constitutional symptoms, as well as severe local inflammation, were produced by applying a mixture of arsenic and soft soap to the pubes and axilla for the purpose of destroying pediculi.[79]

159. Symptoms of acute poisoning.— For convenience of reference, the following brief summary of the usual symptoms occurring in acute arsenical poisoning is given: It will be seen by the above cases and by the detailed reports given in the Appendix, that we may divide cases of acute poisoning by arsenic into two classes, according as death takes place rapidly within a few hours, or, as is most common, is de-

[76-7] Berichte d. d. Chem. Gesellschaft, XII. 2199; also in Archiv. f. exper. Path. u. Pharm. 200-230.

[78] Wiener medizinische Wochenschrift.

[79] Med. Times and Gazette, London, July, 1854, p. 66.

layed for twenty or more hours. In the first class of cases, which usually occur after the taking of a large amount of arsenic upon an empty stomach, the symptoms begin quickly with a burning sensation in the throat, gullet, and stomach, which is quickly followed by nausea and vomiting. There is great thirst, intense pain in the pit of the stomach, soon followed by diarrhea and painful discharges which are usually watery, sometimes resembling the rice-water discharges of cholera, and sometimes they are bloody like the discharges of dysentery. There is usually intense headache; the limbs are cold; the pulse becomes nearly imperceptible; prostration is very great; there is repeated fainting; and no urine, or only a very small quantity, is passed; the expression of the face shows great anxiety; the lips, and often the skin, become blue and livid; convulsive twitchings of the arms and legs occur, and death takes place within twenty-four hours after taking the poison, often in from five to ten hours. It is this form of poisoning which may be mistaken for Asiatic cholera,—especially during an epidemic of that disease. The main points of difference in the symptoms of the two diseases are the burning pain in the throat and gullet, and the fact that in arsenical poisoning the vomiting takes place before the purging; chemical analysis of the urine passed during life or of the organs after death must, of course, be resorted to for confirmation of the diagnosis. In very rare cases of this most acute form of poisoning; like that recorded by Maschka (see Appendix, § 851), the symptoms assume the narcotic form, and pain, vomiting, and purging may be entirely or partially absent, death taking place within a few hours without acute pain.

The shortest fatal period in acute poisoning by arsenic is recorded by Taylor in the case of a young man who died with tetanic convulsions in twenty minutes after the beginning of the symptoms. Recorded cases show no relationship between the amount of poison taken, and the rapidity of the symptoms and fatal period, these depending upon other conditions, such as the condition of the stomach, solubility of the preparation, etc. The period at which death supervenes cannot be definitely stated with reference either to these cases or to those of acute poisoning. In the latter it may take place within twenty-four hours; it may be postponed for several weeks or months. The average period in twenty-two cases, reported by Dr. Geoghegan, was seventy-seven and a half hours, the shortest was five and a half hours.[80] Cases are, however, known, in which death has taken place

[80] Dublin Quarterly Jour. Feb. 1851.

within two hours;[81] and, in the case above mentioned, in twenty minutes.

160. Symptoms of subacute poisoning.— In by far the majority of the cases of arsenical poisoning, however, the progress of the case is much less rapid, the life of the patient being prolonged in many cases for several days after taking the poison, so that some authors have preferred to call this class of cases subacute, rather than acute. In these cases the vomiting which occurs at the beginning of the attack is abundant, frequent, and continues from twenty-four to thirty-six or forty-eight hours, when it ceases for a short period, varying in different cases; during this remission there may be no stomach symptoms, although the sensation of heat in the throat and gullet, the great weakness, excessive thirst, irregular action of the heart and coldness of the limbs remain, and often also the burning pain through the entire length of the intestines with diarrhea and painful discharges. In some cases there is so much pain in the throat and gullet that it is very difficult for the patient to swallow. In a longer or shorter time, according to circumstances, reaction occurs and the symptoms return, often with increased violence, without any fresh dose of the poison having been administered. The abdomen becomes swollen and very tender to the touch, and the general symptoms resemble those mentioned under the first class. In some cases an eruption appears upon the skin, this eruption varying in different cases; sometimes it consists of mere redness, at other times of pimples which may be filled with a watery fluid or with pus. In rare cases the patient has jaundice. In this form of poisoning death usually occurs in from two to eight or ten days.

Those who have partially recovered from the immediate effects of arsenical poisoning, are, moreover, liable to the secondary effects above referred to: salivation, chronic intestinal disorder, palsy, dropsy, and an irritative fever soon prostrate the vital powers; and the fatal termination, although sometimes slowly attained, is, in the majority of cases, none the less certain.

161. Symptoms of chronic poisoning.— When, however, instead of one dose sufficient to destroy life, or several doses at short intervals capable of producing this effect, the poison is given in small portions at comparatively distant intervals, the symptoms are not so marked

[81] The following case will be found in the Lancet, Oct. 1845, by Mr. Iliff: "E. D., the servant in a family, after placing the dinner on the table, retired to her chamber, and drank a glass of water in which she had mingled as much arsenic as it would dissolve; she fell directly and died instantly; no struggling whatever took place."

in their succession, and are attended with phenomena different from those already described. The following may serve as an example: "A woman put daily into the soup of her fellow servant a very small quantity of arsenious acid in powder. Shortly after dinner, this person was seized with vomiting, which led to the rejection of the food and poison before the latter had caused any serious mischief. As this practice was continued for about six weeks, the stomach grew exceedingly irritable; there was pain in the bowels, and the woman became much emaciated. There was also spitting of blood, with such a degree of nervous irritability that a current of air caused an attack of spasms and convulsions. When the patient found that she could not bear anything on her stomach, she left the place and passed two months in the country. Her health became gradually restored there, and she returned to resume her usual occupations. The prisoner, however, renewed her attempts; and, to make sure of destroying her victim, gave her one morning, in coffee, a strong dose of arsenious acid in powder; violent vomiting ensued, and the poison was expelled with the breakfast. Arsenic was detected in the vomited matter, and the explanation of the cause of the long previous illness became clear. Under proper treatment, the patient recovered."[82] Christison relates a case somewhat similar, which, however, was not so protracted, and which terminated fatally.[83] It was by this means, probably, that the crime of secret poisoning was carried, in the seventeenth century, to such a fearful extent. The miserable woman who vended the liquid, called after her "Aqua Tofana," confessed at her death that she had destroyed by it no less than six hundred persons. It is generally supposed that its active ingredient was arsenious acid.

In the above description of chronic, or by some writers called "slow," poisoning by arsenic, it should be noted that this is rather a favorable method used by persons having in mind homicidal or criminal poisoning. The first symptoms in such cases, to arouse the suspicions of a physician, would be shown by the smarting discomfort of the eyelids and eyeballs, conjunctivitis, irritation of the membrane lining the throat and nose, and a peculiar dryness of the tongue and mouth. In this condition the tongue has a red, velvety, coated appearance; the face has a sallow and, sometimes, jaundiced color; the skin is often the seat of an eczematous eruption. There also may be noticed neuralgic pains accompanied with a benumbed sensation in the

[82] Taylor on Poisons, from Flandin, p. 257.

[83] Loc. cit. p. 250, Am. ed.

fingers, with, sometimes, tingling of the toes also, and the muscles are sore on pressure.

The celebrated Maybrick case, in which Mrs. Maybrick was tried and convicted by the Liverpool assizes, 1889, furnishes an example of the above description of chronic or slow poisoning from repeated doses of arsenic administered at intervals during fourteen days. On April 27th, Mr. Maybrick was seized with vomiting after drinking a cup of tea; this continued on the following day, with foulness of the tongue and complaints of stiffness of the muscles of the legs; three days later, he felt unwell after taking luncheon; on the three succeeding days he was sick, complaining of a tickling sensation in the throat accompanied with nausea; ten days after taking the cup of tea first mentioned, diarrhea set in, accompanied with dryness and inflammation of the throat, which, with exacerbations, continued with distressing tenesmus until, on May 11th, he died.

Among other symptoms observed in chronic poisoning, there are catarrhal inflammation of the stomach and intestines, of the nose, of the larynx, and of the bronchi; anemia and various eruptions of the skin; degeneration of the mental faculties, paralysis of the lower extremities, amaurosis, epilepsy, nephritis, inflammation of the liver, and muscular atrophy.

162. Lethal dose.— According to Taylor, Wormly, and Mann, 2 grains is the smallest fatal dose of white arsenic, but Witthaus and Becker fail to find "any record of the clearly established death of an adult from a dose of less than 30 grains (2 gm.) of arsenic in the solid form."

Tardieu mentions, very truly, that this question loses its importance in practice; for, in cases of criminal poisoning or of suicide, it is very rare that the dose of arsenical poison swallowed does not exceed the quantity necessary to cause death, and that it does not attain 5 to 10 or 15 grams (about 75 grains to half an ounce).

The introduction of arsenic into the system by external application (*supra*, § 158) is usually followed by the same alterations in the stomach and intestines as when it has been brought into direct contact with the mucous membrane of these viscera.

In medicinal doses, the solution of the oxid of arsenic produces, sometimes, serious symptoms, and cannot be increased without at once causing symptoms of poisoning. The medicinal dose is from one sixteenth to one twelfth of a grain, and half a grain is sufficient to produce very alarming symptoms. Physicians are accustomed to watch the accession of conjunctivitis, swelling of the eyelids, gas-

trodynia, and general depression, as indications of the dose being too great or too frequently repeated.

Mr. Blyth states that the following may be considered as dangerous doses of arsenic:

2 grains (0.13 gm.) for an adult.
30 grains (1.9 gm.) for a horse.
10 grains (0.64 gm.) for a cow.
½ to 1 grain (0.032 to 0.064 gm.) for a dog.

This writer also states that it has been shown by Gorup-Besanez that, if arsenic acid be mixed with earth, and plants grown in such earth, they only take up infinitesimal quantities of arsenic.

Artificial manure, composed of superphosphates, contains some large quantities of arsenic, and it has been stated that plants take up arsenic from such manure, which is absorbed into their plant tissues.

Kobert states as the lethal dose 15 grains (1 gm.), though he states, also, that generally much more is used. It should not be forgotten that there are historical cases on record, where rectal injections of arsenic have resulted in fatal absorption of the poison.

163. Treatment.— Thorough washing out of the stomach, and the giving of ferrous sulphate in solution freshly prepared, or the ferric hydrate with magnesia (arsenic antidote, U. S. P.):

Solution of ferric sulphate, 50 cubic centimeters.
Magnesia, 10 grams.
Water, a sufficient quantity.

With the directions (U. S. P.) for making this antidote, it is noted "that the diluted solution of ferric sulphate and the mixture of magnesia with water should always be kept (at the pharmacy) on hand, ready for immediate use."

164. Action.— All visible forms of animalcula which live in water die, if a minute quantity of arsenic is dissolved in it. Flies, spiders, and all insects die, if they absorb small portions of arsenic. In worms or leeches, when touched with arsenic acid, the part which is first touched perishes first, and life is extinguished in other parts; their excretions are increased, and vermicular motions, at first lively, become languid, and death gradually takes place. Birds, exposed to the vapor of arsenious acid, become restless, and vomit, and the mucous surface of the nose becomes moist; after which, the respiration becomes labored, and if again submitted to inhale the arsenic acid vapor, they will gradually die in the course of a few days. The symptoms, at first, are loss of appetite, normal breathing, but unusual thirst; finally, the respirations and the heart's action are both slow,

and death gradually takes place very quietly, without convulsions. Examinations of the organs after death show bloodless character of the brain and spinal cord with ecchymoses in the lungs. When the arsenic is administered in solution or in solid form, in a dose less than lethal, the symptoms are languor, loss of appetite, and voidance of liquid excreta like verdigris. After lethal doses, the bird remains quiet; the excreta are large and liquid, sometimes bloody; the motions of the pharynx are spasmodic; the œsophagus shows movements of contraction from below upwards; there is vomiting, general trembling of the body, thirst, feathers erect, labored respiration. The bird then becomes feeble, has convulsions, and dies. All warm-blooded animals, including man, exhibit similar symptoms.

Unlike the mineral acids, arsenic does not corrode dead and living tissues alike. Upon the former there is scarcely any action, while upon the latter the irritation is so great that inflammatory phenomena appear at once, and rapidly destroy the functions of life in these tissues. The skin appears like a burn, and the mucous surfaces become inflamed. The apparent severity of local action of arsenic does not account for the destruction of life, as this latter effect will be exerted mainly upon the nervous centers, the arrest of whose functions seems to cause death of the whole body.

Binz and Schultz state that an animal may be poisoned by dropping a solution of arsenic upon the extremely sensitive surface of the eye (conjunctival membrane), and without causing any serious local irritation, though a slight reddening may follow this local application. The destructive effects upon the internal organs of the body will follow this method of application, even when, apparently, no trace of the arsenic has come directly in contact with their structure; these lesions are observed principally in the stomach. According to these authors, those organic structures or tissues of the body which receive and appropriate oxygen, among which are the glandular structures, are the special seats of destructive action. The neutral salts are just as poisonous as arsenious acid. These authors believe that both arsenic and phosphorus, as well as other reducing agents, act upon the cell growths by discharging their atoms of oxygen, and then become changed into chlorids, and, in this latter form, exert a corrosive action which destroys the cells and blood. As further corroboration of their views, they bring forward the fact that this rapid change by oxidation has been proved in the case of antimony, bismuth, and vanadium. The cellular formation of the various organs concerned with the maintenance of life undergo a fatty degeneration; this converting of the ac-

tive cell formation into fat cells is shown in all of the important organs, such as the heart, liver, and kidneys. Binz[84] has proposed a theory, unproved as yet, that cell protoplasm yields its oxygen to the arsenious acid, converts it into arsenic acid, and then the arsenic acid is again reduced; in consequence of this double action due to the oxidizing influence of arsenious acid, these degenerations result, according to the theory.

165. Diagnosis.— The resemblance between the action of arsenic, locally and in general, to the effect of poisoning from antimony has been previously mentioned. The symptomatic effects of the poisoning are very similar. The similar symptoms: Inflammation of the mucous membrane of the mouth, vomiting and purging, dizziness and prostration, weakness of the pulse, muscular tremors, pustular eruptions, cholera-like and rice-water diarrhea. Differential symptoms, not particularly accentuated for arsenical poisoning: In antimonial poisoning, headache and cyanosis and general paralysis; in arsenical poisoning, pain in the limbs and less obstinate vomiting.

The differential symptoms between arsenical and antimonial poisoning in their chronic forms is rather more accentuated; for instance, conjunctivitis, amaurosis, the dulling of the mental faculties, are more evident in antimonial poisoning, neuralgic pains are more prominent in arsenical poisoning. To assist in the definite diagnosis as between these two poisons, the chemist should be called in, whose isolation of either of these poisons would prove its presence.

166. Post-mortem appearances.— The principal reliable and tolerably constant changes produced by arsenic in the healthy appearance of the viscera are found in the stomach and intestines. The mucous membrane of the stomach is usually of a uniform, deep, brownish-red color, sometimes it is ecchymosed in patches, and at others there are spots or streaks of effused blood. These often have the appearance of crusts, and, being of a blackish color, are not unfrequently mistaken for gangrenous patches, and the slight depression under them for ulceration. But neither ulceration nor gangrene is an ordinary result of simple arsenical poisoning; if found, these are probably merely concomitant lesions, dependent upon other causes.[85] Perforation is exceedingly rare. Orfila says that he has never observed it.[86]

[84] In the Lancet for Sept. 1843, it is reported that the body of a man poisoned by arsenic was disinterred one hundred and forty-one days after death. The stomach and intestines were in perfect preservation. About the middle of the small intestine was found a small ulcerated opening, through which some of the white powder was detected, similar to what was found in the stomach, and which proved to be arsenic.

[85] Blyth, op. cit. p. 551.

[86] Med. Leg. Vol. III. 330.

The mucous membrane is also frequently swollen and thickened, possessing in some parts a fungoid appearance, and its structure is more frequently hardened than softened, owing possibly to a chemical union between the arsenic and the albumin. The powder, if the poison has been taken in this form, is often found imbedded between the folds of the mucous membrane, and closely adherent to it in brilliant points, or in white and flaky patches. The matters contained in the stomach are evidently too variable in character to be enumerated, since the ingestion of different liquids, and of the many so-called antidotes which have been given in most cases, naturally destroys the possibility of drawing any useful inference.

In addition to these inflammatory appearances in the stomach and intestines in cases of acute poisoning, evidences of the irritant action of the arsenic are found in other portions of the body,—especially in the kidneys, which are irritated by the poison as it passes through them, and, in some cases, this irritation reaches such an extent that these organs are found much congested.

Dr. Hans Schmaus, to whose text book on pathology we have previously referred in our opening chapter, states: "Arsenic (arsenious acid) makes more intense lesions of the stomach and intestines than phosphorus, and these are apt to be hemorrhagic. As after phosphorus, peptic ulcers may be observed. In the bloody mucus on the stomach walls, and in the ulcers white crystalline (octahedral) deposits of arsenic; and, on ignition, the odor of garlic is evolved. The gastric effects of the poison are very variable. The general effects are the same as for phosphorus; and, after the poison has wholly disappeared from the alimentary canal, it may be recovered chemically from the liver, kidneys, and bones. The cadaver resists putrefaction, and, long after burial, may be found mummified.[87] In many cases the anatomical condition, after poisoning by arsenic, resembles that of Asiatic cholera, with rice-water discharge, marked enteritis, and swelling of the follicles, so that during an epidemic the two are liable to be confused. A condition, similar to acute, yellow atrophy of the liver, follows phosphorus and arsenical poisoning. The essential element is fatty degeneration. Since death occurs earlier than with acute, yellow atrophy (in disease), often in a few seconds, and, at the longest

[87] A case is reported by Medical Examiner Holt, of a person who died on the 10th of Aug. 1887, and whose body was exhumed Jan. 11, 1888, and the tissues examined by a chemist; he found arsenic in the region of the stomach, kidneys, and spleen. This medical examiner reports that the abdominal walls in this case were "like parchment or leather."

at the end of a few days, the liver may be studied in an early stage, when there is no removal of the degenerated tissues, but when excessive fatty changes are beginning the process. The liver is large, icteric, soft, and friable. If death is delayed for a time, the condition reproduces that of yellow atrophy. In all three conditions the heart, the kidneys, and the epithelium of the alimentary tract suffer albuminous and fatty degeneration. With the poisons mentioned the striated muscles present the same change."

Attention has already been directed to cases of acute and subacute fatal poisoning by arsenic (*supra,* §159). In these cases the organs concerned in the maintenance of animal or organic life,—especially the liver, kidneys, heart, and glands of the stomach,—undergo a serious anatomical change, in which the proper, healthy cells, which are essential to the functional activity of these organs, assume a retrograde change or degeneration of structure, whereby they cease to be active. Consequently, the natural organic functions cease, and life is arrested. The post-mortem appearances in many of these cases can only be estimated or understood by a microscopical examination; this will show to an experienced eye that the cell growth has been replaced by degenerated or fatty tissue, the cells themselves being diminished in number or entirely absent. That this fatty degeneration may occur in an organ as the result of disease, and not the consequence of arsenical or other poisoning, is well known to the medical profession, and yet, as may be learned by reference to a previous paragraph, an experienced pathologist can almost always, by post-mortem observation, determine whether the lesion be the consequence of disease or poisoning. Moreover, the chemical examination can settle positively any doubt which may arise. These remarks apply with equal force to the subacute poisoning by phosphorous, ammonia, antimony, or other poisons producing similar post-mortem appearances, which have been above described.

There are recorded a few cases of post-mortem perforations of the walls of the stomach, but these are very exceptional. There has also been observed a staining of the surface of the liver and of the omentum. And one case is reported by Murray,[88] where the stain occurred in the left side of the heart on the endocardium, a bright, yellow stain deposited over a third of its area; this deposit was proved by analysis to be the yellow sulphid of arsenic. Yellow stains have been observed, also, in the intestines of bodies exhumed after arsenical poisoning. This may be either from the bile pigments or the formation of the

[88] The London Lancet, 1892.

sulphid of arsenic. The inflammatory condition of the mucous surface is most usually observed in the duodenum; but when observed in the large intestines, it is most generally found in or near the rectum. It should be remarked that, in reference to the absence of these inflammatory processes on the mucous surface (referring to the different action of arsenic upon living and dead tissues mentioned in the previous paragraph), an explanation is offered that, in sudden death after arsenical poisoning, this irritant substance may have reached only the further portion of the alimentary tract, after the tissues exposed to the action of arsenic have lost their living functions; consequently, as remarked in the above paragraph, the irritant action ordinarily observed upon living tissue would not take place.

The period of time requisite to develop the inflammatory condition of the stomach is altogether a matter of conjecture, since the mucous coat of the stomach has been found inflamed, when death has followed the poisoning at only the short interval of two or three hours; and, on the other hand, where the quantity swallowed and the duration of life have been such as to lead to the natural belief that inflammation would be discovered, the stomach has been found nearly or entirely free of any such morbid change. Indeed, in a few cases the arsenic has been observed in immediate contact with the gastric mucous membrane, probably for the reason just noted above, without any signs of inflammation. Nevertheless, as a general rule, the inflammatory appearances will be found developed in proportion to the protraction of the case. In a case reported by Dr. Letheby, the stomach was of a pale color. Ettmüller reports the case of a girl in whose stomach arsenic was found, and yet neither in it nor in the intestines was there a trace of inflammation.[89] Orfila, in his work upon legal medicine, says: "The existence or nonexistence of cadaveric lesions, the extent and seat of these alterations, can never enable us to affirm that poisoning has taken place, but can only serve to corroborate the conclusions drawn from the symptoms, and the chemical examination of the suspected matters." These remarks are confirmed by observations of Marc and Chaussier. The inflammatory appearances seldom extend further than the duodenum, although sometimes the small intestine and the rectum exhibit evidence of inflammation. In general, there are no other post-mortem changes at all characteristic of this mode of death. The blood is said to be often syrupy in consistence.

[89] Dr. Taylor records two cases, in one of which ulceration of the stomach was seen, when death occurred five hours after taking the poison; and in the other in ten.

The facts relative to the effect of arsenic upon the putrefactive process are of a very contradictory character. A number of cases are quoted by Dr. Christison, which appear to prove a remarkable antiseptic property in arsenic, by which not only the digestive organs, but the whole body, have been preserved from the ordinary changes of putrefaction. There is no doubt of the preservative quality of an arsenical solution over organic textures placed and kept in it, and the experiments made by Klenck upon dogs seem to show that in cases of poisoning, also, this property may be witnessed. This physician poisoned dogs with arsenic, and left them for two months, sometimes buried in a damp cellar, and sometimes unburied in the same place; the flesh and alimentary canal were red and fresh, as if pickled, at the end of this time. Dr. Kelch, of Königsberg, buried the internal organs of a man who had died of arsenic, and whose body had remained without burial till the external parts had begun to decay, and, on examining the stomach and intestines five months after, he found that the hamper in which they were contained was very rotten, but that they had a peculiar smell, quite different from that of putrid bowels, were not yet acted on by putrefaction, but were as fresh as when first taken from the body, and might have served for the purpose of anatomical specimens. The body of Chapman, supposed to have been poisoned by Mina, was disinterred two months after death. The face was livid and putrid, but the odor of the corpse was not offensive. The abdomen was of a pale-white color, and Dr. Hopkinson, on cutting into it, was struck with its firmness and resistance. When the stomach was opened, a very peculiar smell was perceived, which he compares to that of pickled herring. The same remark was made by other medical witnesses.[90] The intestines were entirely empty, of a pale color, and, apparently, rather disposed to dry than to putrefy. In a case communicated by Dr. Traill to Dr. Christison, the body of a captain of a vessel was disinterred five months after death. The face and neck were swollen, black, and decayed, but the rest of the body was quite free from the usual signs of putrefaction. The skin was white and firm, the muscles fresh, the lungs crepitating, the liver and spleen much shriveled, the stomach and intestines entire throughout their whole extent, and capable of being handled freely without injury. In this instance the coffin contained water, owing to its having lain in a sandy soil resting on clay. The remarkable preservation of the body

[90] In two cases of poisoning by arsenic, observed by Dr. Sanborn, of New Hampshire, the same peculiar odor was distinctly observed. Boston Med. and Surg. Jour. Vol. XXXVII.

of a woman, supposed to have been poisoned by arsenic, for nearly fourteen years after her death, led to its disinterment, the indictment and trial of her husband. Arsenic was found in the body.[91]

On the other hand, Dr. Geoghegan has observed examples both of very tardy and of very rapid decomposition in cases of arsenical poisoning.[92] It would not be difficult, also, to find many cases in which, although death has resulted from other causes, the body has been as remarkably preserved as in those where arsenic was the cause of it. We have elsewhere enumerated the various causes which will retard putrefaction, as the dryness of the soil, and the depth at which the body has been interred, as well as individual peculiarities, which do not always admit of explanation. We may quote here an observation which will show that, even under the most favorable circumstances for decomposition, this process may proceed very slowly. This is a case communicated by Dr. Routier, of Amiens, to Orfila, in which an old woman was destroyed by a blow upon the head with an ax. This was in the middle of summer. The body lay buried in the clayey soil of a cellar between eight and nine months. At the end of this time, a judicial inquest was held. The skin was perfect, the muscles firm, red, and distinct, the cerebrum was like that of a fresh corpse, and possessed its natural firmness and proper odor. The viscera of the chest and abdomen were perfectly well preserved; and in the stomach, which presented no signs of inflammation or other disease, a thick fluid was found, in which the remains of articles of food were distinctly recognized.[93] The fact of the remarkable preservation of the brain, in this case, is also of some importance; for it is well known that usually it putrefies rapidly, and because, also, of a case, lately reported by M. Dieu, where the body of a man, poisoned by arsenic, was disinterred after the lapse of two and a half years, the comparative integrity of the brain being attributed to the preservative powers of this substance.

In a summary furnished by Tidy,[94] it is asserted "that arsenic, antimony, chlorid of zinc, also chloroform, phosphorus, and strychnin, when they are actually the cause of death, usually retard decomposition. In arsenical poisoning, putrefaction ordinarily commences as usual, but seems to stop after it has commenced, then a process very similar to mummification begins." And again,[95] "thus arsenic acts

[91] Webster. Boston Med. and Surg. Jour. Vol. XXXIX. p. 489.

[92] Dub. Quart. Jour. Feb. 1851.

[93] Orfila, Traité de Méd. Lég. 4ème ed. Vol. II. p. 93.

[94] Legal Medicine, Phila. 1882, p. 91.

[95] *Ibid.* p. 106.

commonly (but by no means necessarily) as a preservative agent, so that in a case of arsenical poisoning, a fairly accurate opinion may sometimes be formed as to the condition of the mucous membrane, after a lapse of even three or four weeks."

Hence the medical witness cannot be authorized to assert that, because the body has resisted more or less completely the progress of putrefaction, this preservation is due to arsenic, since it may be really attributable to other causes. One cause of the discrepancy in the observations upon this point undoubtedly lies in the variable time occupied by the poison in producing its fatal effect, and the nature of some of the symptoms. It is reasonable to suppose, and observation also has shown, that, if death has resulted from the ingestion of the poison at repeated intervals, in small doses, or not until several days have elapsed, the arsenic has been disseminated through the system, and may thus exercise a more complete antiseptic influence in the dead body. If, again, the person has died within a short period after taking the poison, and after abundant and repeated vomiting and purging, we may be right in anticipating that, although the violent action of the poison has been the cause of death, little or none will have remained in the body. Hence, in the latter case, putrefaction will probably pursue its ordinary course. Thus, in a case examined by Dr. Geoghegan, of a person who died sixteen days after taking a large dose of arsenic by mistake, no trace of the poison could be found in any part of the body.

167. Embalming fluids.— (On imbibition.) Embalming fluids usually contain arsenic, as well as other poisonous articles, and interpose a serious question as to the discovery and isolation of arsenic in a suspected case of poisoning. In the trial of Sarah Jane Robinson this became an important point in the defense. We have previously referred to this trial and the official report of the testimony (see page 31). Among the first of her supposed victims was her sister, who died on the 26th day of February, 1885, and for whose death no bill of indictment was presented. Before the body was cooled, it was given in charge of the undertaker, who found the corpse distended with gas to such an extent that he tapped the abdominal wall to let off the gas; as the swelling went down, he injected considerably less than a pint of embalming fluid; he admitted that most embalming fluids contain arsenic. It is interesting to note, in this connection, the testimony of the chemical experts. Prof. Hills could not be made to say, in his testimony, that, in an embalmed body, the presence of arsenic, when found, was indicative of having been administered before death.

While, *per contra,* Prof. Wood testified that on account of its unequal distribution throughout the various tissues of the body, it might be inferred that the arsenic recovered from an embalmed body was injected post-mortem; and that, if the amount of arsenic found in the body was uniformly distributed throughout its organs, the inference might be drawn that the poison was received and absorbed before death. In other words, the effect of embalming would be distribution by a slow and unequal diffusion after death, when injected into a dead body; while, if administered during life, it would pass into the tissues of the organs by the living processes of absorption.

It has sometimes been contended in trials that a buried body may imbibe arsenic from the surrounding arsenical soil in a cemetery or graveyard; when arsenic is found in such soil, it is always in an insoluble form combined with iron, as arsenical iron pyrites. In his testimony before the court at the above Robinson trial, Prof. Hills gave his opinion that, from recent observations, bodies lying in arsenical soil do not imbibe arsenic in their dead tissues. Where bodies are exhumed for chemical examination for arsenic, it is always advisable to collect samples of the soil surrounding the corpse, for the purpose of analysis.

168. Chronic poisoning from absorption in factories, wall papers, pigments, cloth textures, etc., where arsenic is used.— Chronic poisoning is caused by the absorption of small amounts of arsenic, continued for a long period. This is sometimes due to taking some preparation of arsenic as a medicine, but far more frequently by its use as a pigment in ornamenting articles of household use, such as wall paper, articles of clothing, toys, water-color paints, wrapping and other papers, and sometimes, even, it accidentally gets into articles of food and drink, such as confectionery and wines or syrups. The principal preparations of arsenic which are the sources of this form of poisoning are the arsenites of copper, the two sulphids of arsenic, and arsenical anilins. The persons most liable to this form of poisoning are workmen engaged in various manufactures in which these pigments are used,—especially wall paper and artificial flowers,—and those whose occupation is to place these papers on the wall. Two cases, one of which was fatal, are reported by Hoffmann and Ludwig.[96] A woman sixty-three years of age, and her daughter twenty-two years of age, were engaged in making crosses from artificial moss and flowers, a very large number being made during the few weeks before All Souls' Day. The first year both were poisoned, but the poisoning

[96] Wien. Med. Jahrb. 1877, p. 501.

was only temporary; on the second, however, the symptoms were very severe, the daughter recovering after several months, but the mother died. After death, extreme fatty degeneration of the liver, kidneys, heart, and muscles was found, and chemical analysis showed the presence of arsenic in all of the tissues except the bones, the largest amount being found in the brain and liver. The source of the poisoning was traced to the arsenical fuchsin used for coloring the artificial flowers; of six different kinds of fuchsin found in the shops, only one was found free from arsenic.

It is not usual for a workman to be employed so continuously with arsenical pigments that a directly fatal result ensues; he usually suffers only with a more or less severe irritation of the skin and mucous membranes with which the pigment comes in contact, such as inflammation of the nose (coryza) and eyes (conjunctivitis); if much of the dust gets into the mouth, there is usually a sore throat and, perhaps, some irritation of the stomach, which is quite certain to occur if the exposure be long continued. Those portions of the skin which are habitually moist with perspiration, as in the folds of the groin, about the scrotum, and in the armpits, are especially apt to be inflamed; or, if any portion of the skin be kept moistened or continually exposed to the action of the arsenical pigment for some time, it will become inflamed and covered with an eruption. If the exposure be continued sufficiently long, there will follow, next, evidences of irritation of the stomach and intestines, as shown by more or less dyspepsia, loss of appetite, perhaps nausea and vomiting, and looseness of the bowels; still later, various nervous symptoms appear, consisting chiefly of neuralgic pains in various portions of the body, and often more or less hysteria.

The number of sources of this form of poisoning is very great, but the source of the greatest number of cases is the use of arsenical pigments in wall paper; persons occupying rooms, the walls of which are covered with such paper, being liable to symptoms very similar to those above described in the case of workmen; but often, especially in the case of women and children, who are almost constantly exposed to the action of such paper, symptoms of poisoning are more apt to occur than in men, or in workmen engaged in the manufacture of such paper (unless the amount of arsenic is very large), both on account of the greater susceptibility of women and children to its influence, and on account of the greater constancy of the exposure,—especially in chambers badly ventilated or used for sleeping apartments. Formerly, only green papers were suspected to contain arsenic, it being

in the form of one of the compounds of copper and arsenic, but of late years, probably owing largely to the popular suspicion in regard to bright green papers, the number of arsenical greens has diminished, and we find a very large number of arsenical reds, pinks, browns, and blues; in fact, nothing whatever in regard to the arsenical or non-arsenical nature of a wall paper can be inferred from its color. The amount of arsenic which some wall papers contain is very large, varying from a mere trace to 30 or 40 grains to the square foot. In one case of red-colored wall paper, the color contained 2.49 per cent of arsenic. The number of authentic records of cases of chronic poisoning from this source, some of which have proved fatal,[97] is so great that there can be no possible doubt about it.

In one case of illness traced to poisoning by arsenical wall papers,[98] from a careful analysis of excreta from the patient, and dust from the bedroom, which was decorated with arsenical paper, the following results were obtained:—

	White arsenic.	Scheele's green.
100 grains of dust from top of wardrobe contained	0.2	.36
100 " " " same room.....	0.16	.30
Expectoration from patient.....	trace	trace
48 ounces of patient's urine.....	0.26	.50

The next most common sources of this form of poisoning are articles of dress, such as artificial flowers and cloth used for dresses; in artificial flowers, both the green leaves may be highly arsenical from the presence of Paris green, and the flowers, from the presence of arsenical fuchsin, as in the cases mentioned above. A lady was prostrated with severe symptoms of arsenical poisoning by wearing a dress made from a fabric which contained about one half a grain of arsenic to the square foot (0.291 gm. per square meter); this was a specimen of cloth, known in the shops as "foulard cambric," of dark and light blue stripes. A specimen of garnet woolen brocade, which was intended for use as a bed-hanging for an infant's bed, was found highly arsenical. The most numerous cases recorded of articles of dress being the source of chronic arsenical poisoning are due to green tarlatan, which has produced severe symptoms in a large number of cases. The way in which arsenic may get into cloth may be seen by a glance at the following recipes:—[99]

[97] See Med. Times and Gazette, May 3, 1862, and Appendix.

[98] Brit. Med. Jour. June 21, 1873.

[99] Dyeing and Calico Printing by the late Dr. F. Crace-Calvert, F. R. S., F. C. S., 1876, p. 73.

Mordant for madder purple.—

"For light purple:

1 gallon of acetate of iron at 1½° T.
3 gallons of purple assistant liquor.
3 gallons of water.
18 pounds roasted farina.

This solution is ready for use after straining.

"For dark purple:

7 gallons acetate of iron at 2½° T.
¾ gallon purple assistant liquor.
3 pounds flour.

"The purple assistant liquor is made as follows:

1st. 100 gallons of wood acid.
9 pounds yellow prussiate of potash are dissolved in the wood acid.
2 gallons of sulphate of lead precipitate; made by dissolving acetate of lead in water, precipitating with sulphuric acid, and allowing the sulphate of lead to subside.

2d. 50 pounds carbonate of soda dissolved in 8 gallons of water, to which is added 50 pounds arsenious acid; when this is dissolved, it is added to the first mixture, and thoroughly incorporated by stirring.

To the above complete mixture, add 3 gallons of hydrochloric acid and 200 pounds of common salt. Mix well and store for use."

"Printing with madder extract.[100]—

"For dark purple:

Boil together 1 liter of extract (Pernod's) in paste.
1 liter of acetic acid at 8° Baumé.
224 grams of olive oil.

The acid which is evaporated is replaced, and the mixture is thickened with 600 grams of powdered gum senegal. When required for use there is added—

224 grams of black mordant at 10° Baumé.
128 grams of arsenite of soda at 6° Baumé."

The following is an extract from the formula for making magenta:[1] "Into this apparatus, which is capable of holding about 500 gallons, a charge consisting of 2740 pounds of a concentrated solution

[100] *Ibid.* p. 97.

[1] *Ibid.* p. 366.

of arsenic acid, containing 72 per cent of the anhydrous acid, is introduced, together with 1600 pounds of commercial anilin."

There are many other sources of chronic arsenical poisoning, as, for instance, anything which is habitually handled or eaten, if colored with an arsenical coloring matter. The most important of these are children's playthings, which are not only handled, but are often sucked by the child; toy paints,—especially light green,—the coloring matter of which is often pure Paris green; confectionery, both green and red, of which there has been a fatal case recorded;[2] artificially colored syrups, vinegar, jellies, and wines; the use of such pigments in the manufacture of wrapping paper and glazed papers, so often seen on paper boxes, and as wrapping paper for lozenges; these papers are also largely used by children in kindergarten schools, where they may do a great deal of harm. Of six specimens of these papers examined for the writer by Dr. C. Harrington, one of the reds (unglazed) was found to contain about 1 grain of white arsenic to the square foot (0.690 gm. to the square meter); another red (glazed), about one sixth of a grain to the square foot (0.096 gm. per square meter); one of the green (glazed), about seven and one fourth grains to the square foot (4.341 gm. per square meter); another green (glazed), about three grains to the square foot (1.76 gm. per square meter); an unglazed green about five and one fourth grains to the square foot (3.162 gm. per square meter); and a second unglazed green about three and one fourth grains to the square foot (2.626 gm. per square meter). A child was given a green card to play with, and soon afterwards was seized with the symptoms of arsenical poisoning. It was found that the glazing of the card, which contained lead, was colored with Scheele's green.[3]

Arsenic in wall paper does not act merely by virtue of the dust, which becomes detached from the paper, and diffused throughout the air of the room, so that it comes in contact with the mucous membranes, but it also acts by becoming partially converted into the exceedingly poisonous gas, which is formed when any of these arsenical pigments remain in contact with moist organic matter, or from an organic combination of arsenic oxid (*supra*, § 168; also Appendix, case No. VIII.)

In reference to accidental absorption of poison from arsenic used in the arts, including wall-papered rooms, we again refer to the record of the Robinson trial.[4-5] The defense attempted to show that the

[2] Jour. de Chimie Médicale, 1875, p. 131.
[3] Boston Med. and Surg. Jour. Vol. XXXVII. p. 107.
[4-5] Appendix, Case No. VIII.

contention of the government, in one of the cases adduced to prove the motive of the accused to be deliberate action to poison her victims, was ill founded; even if arsenic had been found to have been administered before death, the poison might have been received and absorbed by the individuals from the vapor of arsenic in iron works where the man was employed, or from the vapor emanating from wall papers containing arsenic, either as a dye or a mordant. As for the former being the origin of the source of the poison, it was shown by the chemical expert that there was not enough arsenic used in the processes of the iron works to account for the fatal dose; as for the wall paper furnishing the arsenic, Prof. Wood stated in his cross-examination that he knew of two cases of poisoning from arsenical wall paper, to which actual poisoning might reasonably be attributed. These two cases were under the peculiar condition that there was a large amount of arsenic present, a great many grains to each square yard of the paper; and that, in the apartments where this wall paper was attached, the air was confined and moist for a long period of time; this would predispose to the evolution of sulphohydrate of arsenic. This is a form of arsenical vapor to which accidental, arsenic poisoning is attributed. He knew of no other cases of accidental, arsenical poisoning, except when the arsenic was heated.

Arsenic, formerly, was one of the ingredients contained in candles, for the purpose of hardening the wax and to give a glossy effect; of course, when lighted, these candles would vitiate the atmosphere with arsenic, in a room in which they were used. Weed destroyers frequently contain arsenic among the ingredients composing them. Solutions containing carbonate of soda, for the purpose of cleaning out boiler tubes, have, sometimes, arsenic contained in them,—as much as 40 parts of white arsenic to 9 parts of the carbonate of soda; when used in connection with steam for scouring these tubes, arsenical vapors might be given off. Arsenic has also been employed for surfacing note paper, cardboard boxes, playing cards, and wrappers for sweetmeats and ice cream. Arsenic has also been found in the glazed, leather linings of hats, and thus, by contact with the skin of the forehead, eruptions have occurred.

Where persons are employed in factories where arsenical vapors are evolved into the air, arsenic might be absorbed from inhalation by the lungs and, perhaps, by swallowing the saliva, and so passing into the system, produce symptoms of chronic poisoning such as headache; neuralgia or neuritis; inappetence; gastric distress; irritation of the throat, eyes, nostrils; conjunctiva inflamed; eyes intolerant to light;

gradual prostration; anemia; unhealthy hue of the skin; and disturbances connected with the nervous system such as trembling of the muscles, and obstruction in locomotor action, and in response to reflex external irritation. In fact, these symptoms somewhat resemble those of chronic alcoholism. The motor symptoms are the result of muscular atrophy and a degeneration of the muscular tissues. The reflex symptoms are shown by absence of the knee jerks. These symptoms may also be caused by the long continuance of medicine containing arsenic, as reported by Prof. Osler.[6] In all these cases of chronic poisoning from accidental absorption of arsenic, this poison should be found in the urine by chemical tests.

169. Chemical examination.—Arsenious acid, in its chemical relations, must be considered 1st, as a solid; 2d, in solution; 3d, mixed with organic matter.

When a portion of the solid is thrown upon the surface of water in a vessel, only a small part sinks to the bottom, most of it floating upon the surface, although much heavier than water; this serves as a ready preliminary test to distinguish arsenic from other heavy, white, crystalline powders, such as corrosive sublimate, calomel, lead, bismuth salts, etc. It is entirely volatilized by heat, a temperature of only about 218° C. being required. This is also a ready preliminary test which will enable the physician, even at the bedside, to distinguish it from other mineral poisons, except corrosive sublimate and calomel, since it is only necessary to heat a little upon a knife blade over a common lamp or gas flame. This test is better performed by heating a little of the powder in a glass tube closed at one end, such as a small test tube, when the powder will disappear from the heated end of the tube, but will be condensed upon the cool portion in the form of a white, crystalline sublimate, the crystals having the octahedral form, as can be ascertained by examination with an ordinary pocket lens. Its solubility has been mentioned above (*supra*, § 156).

Thrown upon ignited charcoal, it gives off an alliaceous odor, due to the reduction of arsenious acid, which has in itself no odor when heated; the smell of garlic is perceived only when it is deoxidized. This odor, although striking, is not a positive proof of the presence of arsenic, as a similar one may be given off by several other substances. If, however, we heat arsenious acid with dry acetate of potassium, oxid of cacodyl is disengaged, by the peculiar insupportable

[6] Montreal Med. Jour. 1893.

smell of which compound, even very minute traces of arsenious acid may be detected. This experiment may be conveniently performed by rubbing the substances together in a little mortar, and then heating them together in a test tube, allowing the vapor, which is excessively poisonous, to be carried away by a current of air.

Heated in a narrow test tube, or in the reduction tube of Berzelius, with some freshly ignited (coal) charcoal, the same phenomena of deoxidation and evolution of odor occur as when it is placed on red-hot cinders in the open air; but, in this case, metallic arsenic is condensed by sublimation upon a cool portion of the tube, in the form of a metallic crust or ring, technically called an arsenical "mirror," of an iron-gray color, brilliant and lustrous upon the outer surface, and crystalline upon the inner, when seen under a low magnifying power. There are usually two crusts deposited, an upper and a lower, the latter of metallic arsenic, and the other of a browner color, which is a mixture of the metal and its oxid.

The arsenical nature of the ring may be further proved by volatilizing it by heat, in an open tube, by means of the flame of a spirit lamp; it is thus converted into arsenious acid, which sublimes upon the tube in the form of octahedral crystals, which may be dissolved in distilled water, and subjected to the liquid tests; or, by dissolving them by means of nitric acid, and evaporating the solution to dryness, arsenic acid is formed, which is known by its giving a brownish-red precipitate, with nitrate of silver. When the quantity of arsenic is considerable, it is better to use a flux composed of the residue left by tartrate and acetate of sodium after incineration in a covered platinum crucible,—black flux, as recommended by Dr. Taylor. The volatilization of the crust may be accomplished either by applying heat directly to it, and chasing it up and down in the tube until it is all oxidized; or, by carefully filing off that part of the tube in which it is contained, powdering it, and then introducing it into the end of another tube, which should in turn be subjected to heat; or, still better, by breaking off the sealed end, and, when heating the mirror, slightly inclining the tube so as to insure a current of air through it, when the white crystalline sublimate of arsenious oxid will be seen a short distance above the point at which the flame was applied.

The principal precaution to be taken in performing this test is to be sure that all of the substances used are perfectly dry. The metallic crust of arsenic may, moreover, be distinguished from the discoloration produced by charcoal, by the absence, in the dark stain of the

latter, of any metallic appearance; the inner surface of the charcoal discoloration being powdery, black, and dull. This test is called the reduction test for arsenic, and is especially valuable because it can be applied to any of the solid compounds of arsenic, whether soluble or insoluble; and also, because, when properly and delicately performed, a satisfactory mirror can be obtained with an amount of arsenical compound corresponding to only 1/1000 part of a grain of white arsenic,[7] and also, especially because this test does not apply to compounds of antimony, which are so liable to interfere with many of the tests for arsenic.

The sublimate obtained by the reduction of the compounds of mercury, as calomel or corrosive sublimate, has indeed a metallic appearance, but may be distinguished, without using the liquid tests, by an inspection with a common lens, or even with the eye; the minute globules of metallic mercury can thus be readily seen, and by the point of a knife or a glass rod be made to run together.

The objections that have sometimes been made to the reduction process, on the ground that glass contains arsenic or lead, are theoretical only. Glass does not contain arsenic; for, although used in its manufacture, it is entirely volatilized by the heat required in the process, and when the glass does really contain lead (which ought not to be the case in suitable chemical implements), the mere loss of transparency caused thereby, upon the application of heat, is evidently in the substance of the glass itself, and cannot, with the slightest attention, be mistaken for the arsenical crust. Moreover, the discoloration caused by the reduction of lead will be in the part of the tube to which the flame is applied. A crust weighing only 1/300 of a grain, 1/10 of an inch broad, and 4 times as long, may show, characteristically, all the physical characters of an arsenical sublimate 100 times larger. It may therefore be safely laid down that the appearances, exhibited by a well-formed, arsenical crust contained in the minute quantity of 1/300 of a grain, are imitated by no substance in nature which can be sublimed by the process for the reduction of arsenic.[8]

If either white arsenic or metallic arsenic, as obtained by the reduction test, be treated with concentrated nitric acid, and the mixture evaporated to dryness, the residue consists of arsenic acid, which can be dissolved in water and the appropriate tests applied (see below). The same result can be obtained by treating the white

[7] Wormley, Micro-chemistry of Poisons, 1885, p. 259.

[8] Christison.

arsenic or metallic arsenic with concentrated hydrochloric acid, adding a few crystals of potassium chlorate and evaporating to dryness. The sulphid of arsenic can also be oxidized by these last reagents, to the form of arsenic acid.

The liquid tests for the detection of arsenic in solution are several in number. They are applied to clear solutions of arsenious acid free from organic matter, and are extremely useful in corroboration of the tests by which it is obtained in a metallic state; the arsenical deposit having first been converted into arsenious acid by sublimation in an open tube, and then dissolved in water.

170. Hydrosulphuric acid test.—In the presence of free acid (hydrochloric), hydrosulphuric acid (sulphureted hydrogen) throws down the trisulphid of arsenic, which is of a lemon-yellow color. This precipitate is soluble in ammonia, sulphid of ammonium, and the fixed alkaline hydrates, by which means it can be separated from the sulphids of mercury, lead, copper, bismuth, and cadmium, if it happens to be mixed with these compounds. It can be converted into arsenic acid by hot nitric acid or hydrochloric acid and potassium chlorate, and the tests for arsenic acid obtained with the resulting fluid. Dried, and heated with black flux in a reduction tube, a metallic sublimate of arsenic may be obtained (reduction test).

171. Ammonio-nitrate of silver test.—This reagent should be carefully prepared. (To a strong solution of nitrate of silver, add a dilute solution of ammonia, until the brown hydrate of silver, which is thrown down, is nearly, but not entirely, redissolved. When properly prepared, there should be no free ammonia given off.) The arsenite of silver, which is precipitated by this reagent, is of a lemon-yellow color, which is soluble both in ammonia and nitric acid. It must be noticed that phosphate of sodium also produces, with nitrate of silver, a similar precipitate, which is equally soluble in nitric acid and ammonia.[9]

It frequently happens that common salt or hydrochloric acid is present in the solution, together with the arsenic; the chlorin must first be removed before we can see the lemon-yellow color of the arsenite of silver; this is done by adding a drop or two of nitric acid to the solution, and then an excess of nitrate of silver solution, and shaking; the chlorin will thus be entirely precipitated in the form of chlorid of silver, while the arsenite of silver will remain dissolved by the nitric acid, and can be separated from the chlorid

[9] Bowman and Bloxam, London, 1871.

by filtering; upon exactly neutralizing the filtrate with ammonia, the yellow arsenite of silver will be precipitated.

172. Ammonio-sulphate of copper test.— (This reagent is prepared in the same manner as the foregoing. No more than is actually necessary for precipitation should be used, as its intense blue color is very apt to mask the proper color of the precipitate. If, however, this has occurred, filtration will separate the green arsenite of copper from the uncombined portion of the liquid.) The color of the precipitate is a chrome green. This precipitate is the familiar Paris green. It is readily soluble in an excess of ammonia, to form an azure blue solution, and in free acids. When dried, and heated in a test tube, arsenious acid is disengaged, and sublimes on the sides of the tube in the characteristic crystals, leaving a residue of the oxid of copper. Their nature may further be proved by dissolving them in distilled water, and submitting them to any other of the tests which may be desired. In relation to this test, it must also be borne in mind that a similar precipitate is produced, when the solution of copper is added to liquids containing some vegetable substances, though no arsenic may be present.[10]

173. Objections to these tests.— The fallacies, to which these tests are exposed, are the following: Phosphoric acid gives a yellow precipitate with ammonio-nitrate of silver, exactly like arsenious acid; several organic acids cause a green precipitate with the copper test; and the soluble salts of cadmium yield, with sulphureted hydrogen or hydrosulphuric acid, a yellow precipitate, similar in appearance to the sulphid of arsenic. But doubts, arising from these sources, as to the true character of the precipitate, may be corrected by a comparison of the tests, and the production of a metallic or crystalline sublimate. Thus, phosphoric acid gives only a pale blue precipitate with the copper test, and is not affected by sulphureted hydrogen; and the pseudo-arsenical precipitate, obtained by cadmium with sulphureted hydrogen, or by organic acids with the copper test, when dried and heated in a reduction tube, gives neither the metallic deposit, such as is obtained from the sulphid of arsenic, nor the crystalline sublimate as obtained from the arsenite of copper. "No chemist in the present day would think of employing these liquid tests in solutions in which arsenic was mixed with organic matter. Almost all liquids, used as articles of food, are precipitated or colored by one or both of them, somewhat like a solution of arsenic, although none of this poison is

[10] *Ibid.* p. 82.

present. An exclusive reliance upon them, as color tests, has led to the rejection of chemical evidence on several trials."[11]

If a solution of arsenic be rendered strongly alkaline with potassium or sodium hydrates, a few drops of solution of sulphate of copper added, and the mixture boiled, a yellow precipitate of the suboxid of copper will be formed, owing to the removal of a half of the oxygen from the oxid of copper by the boiling, alkaline solution of arsenious acid, which is itself converted into arsenic acid. This reaction is also caused by other reducing agents, especially grape sugar.

174. Marsh's test.— This process for obtaining arsenic from simple or compound mixtures, by which it may be afterwards tried by any or all of the tests above mentioned, is exceedingly delicate. According to Dr. Christison, a solution, containing only a millionth part of white arsenic, will part with it readily in the form of arseniureted hydrogen, and the slightest trace of that gas in the hydrogen is indicated by this method. The process consists, essentially, in the disengagement of hydrogen gas by the action of sulphuric acid on zinc, and, in the presence of arsenious or arsenic acids, the consequent evolution of arseniureted hydrogen, and the deposition of metallic arsenic upon a porcelain plate held in the flame, resulting from the combustion of the gas, or within the glass tube through which the gas is passing, the gas being decomposed by a flame applied to the tube.

The various modifications of the simple apparatus of Marsh require no particular description here. The simplest form consists of a wide-mouthed bottle with a closely-fitting cork perforated for two tubes, of which, the one, furnished with a funnel, dips beneath the liquid, and the other, bent nearly at right angles but sloping slightly towards the bottle, descends but a short distance into the vessel. This tube should be connected with some apparatus for drying the gas, such as a large tube filled with pieces of fused chlorid of calcium, or a bottle or flask containing a little concentrated sulphuric acid. The gas, after being thus dried, is made to pass through a hard, glass tube, which is drawn out to a fine point. In this apparatus, hydrogen is generated by pure zinc and dilute sulphuric acid, and the action is continued until the atmospheric air is completely expelled before lighting the escaping gas, and all risk of an explosion is thereby avoided. The freedom from arsenic of the materials employed should be ascertained by applying a flame to the exit tube for half an hour; if, at the expiration of that time, no mirror of

[11] Taylor on Poisons, 3d Am. ed. 1875, 309.

metallic arsenic has been deposited upon the inner surface of the tube, we may be sure that the materials employed are absolutely free from arsenic. In this country there is usually no difficulty in obtaining zinc which is perfectly free from arsenic. When thus satisfied that the materials are pure, a portion of the suspected liquid may be poured into the funnel tube, and the spirit lamp be immediately applied to the horizontal tube, in order to obtain a metallic ring or incrustation, which, if arsenious acid is present, will be deposited at the distance of about half an inch from the part to which the flame is applied. Having procured this, the gas, as it issues from the fine end of the tube, should be ignited, and deposits obtained on porcelain or glass. The two processes may be continued until a metallic deposit is no longer obtained.

These rings or mirrors, as they are termed, should be tested to ascertain their arsenical nature as follows: That portion of the tube containing one of them should be filed off, and gently heated while the tube is slightly inclined so as to insure the passage of a slow current of air through it; the dark ring will gradually disappear, and a short distance above, on the cool portion of the tube, will appear a white crystalline sublimate of arsenious oxid, which can be tested by the appropriate tests. Another portion of the tube, containing a mirror, should be attached to a sulphureted hydrogen apparatus, and the mirror gently heated while a very slow current of sulphureted hydrogen is passing through the tube; the dark ring will disappear, as when heated in a current of air, and there will appear at a short distance from the flame a lemon-yellow sublimate of sulphid of arsenic, which will be found to be insoluble in hydrochloric acid, but soluble in sulphid of ammonium. The mirror in still another portion of the tube should be touched with a solution of sodium hypochlorite, which will immediately dissolve it (distinction from antimony mirror formed in the same way). The mirror in another portion of the tube should be touched with a solution of sulphid of ammonium; it should remain undissolved, whereas the corresponding antimony mirror would be quickly dissolved. Of these substances, the three tubes containing the arsenical mirror, the sublimate of arsenious oxid, and the sublimate of the yellow sulphid of arsenic, should be taken to court in legal cases as *corpora delicti*. The mirrors, obtained upon the porcelain plates by depressing them into the ignited flame at the end of the tube, should also be tested as to their arsenical nature. One should be touched with a solution of sodium hypochlorite, in which, if arsenic, it will quickly dissolve. Another will be found to

be insoluble in a solution of sulphid of ammonium. Another should be dissolved in a drop of nitric acid, the solution gently evaporated to dryness, and the residue, which will consist of arsenic acid, touched with a drop of a solution of ammonio-nitrate of silver, when the brick-red arseniate of silver will be seen.

If a cold beaker or other glass vessel be held over the flame of arseniureted hydrogen burning at the end of the tube, moisture will condense upon the inner surface of the glass, which, after drying will contain a white residue consisting of white arsenic, the arseniureted hydrogen, on burning, being converted into water and white arsenic. Marsh's test cannot be successfully performed, as a rule, with solutions which contain organic matter; this should therefore be first removed by the methods mentioned below.

175. Objections to Marsh's test.— The fallacies, to which Marsh's process may give rise, proceed from the contamination of the zinc or sulphuric acid with arsenic, the presence of antimony in the suspected liquid, or of imperfectly charred organic matter. The mode of guarding against the first has already been noticed. Antimoniureted hydrogen burns with a pale, bluish-green flame, and deposits a black stain upon a porcelain plate held in it.

The antimonial is distinguished from the arsenical crust by the following characters:

First. The dark stain is less bright and metallic than the arsenical one; and, when viewed by transmitted light, is smoky-black, whereas, that of arsenic is hair-brown.

Second. If the flame be allowed to play on a solution of ammonio-nitrate of silver, placed on the under surface of a plate of mica, no yellow arsenite of silver is obtained.

Third. The greater volatility of arsenic, and its conversion into octahedral crystals of arsenious acid, may serve, where the crust is in an open tube, to distinguish it from antimony. This may be effected by a bath of olive oil; this liquid does not begin to boil until the heat rises above 600° F. Arsenic is completely sublimed under 500° F., and the process begins at a much lower temperature; but antimony is not at all affected by the heat required to boil olive oil. Hence, whether the stains of the two metals are mixed or not, their true nature can thus be readily ascertained. Dr. Maclagan says that in his hands the process has proved "so simple and easy of execution, so delicate in the results obtained by it, so advantageous in excluding the necessity of any chemical reagent whatever," and "also in affording, when tubes of equal size are used, so easy a method of determin-

ing, approximately, the proportion of arsenic in different articles examined, that in operating on small quantities of material, or where little arsenic is present, I have of late always, in practice, adopted it in preference to any other."[12]

Fourth. The tests applied to the metallic deposits inside of the tube mentioned above.

Fifth. The metallic crust, obtained by submitting a current of the gas to heat, presents some distinguishing characters; the arsenical crust is always deposited in the more distant or anterior part of the tube, whereas, the antimonial one is first deposited on the heated part of the tube.[13]

Sixth. Arsenical spots on porcelain may also be readily distinguished from those of antimony by the more rapid solution of the former in hypochlorite of sodium. They are rapidly dissolved by it, and the porcelain becomes perfectly clean. If they are shining and thick, the process is somewhat longer, but does not occupy more than a few seconds. Antimonial spots, on the contrary, completely resist the action of the hypochlorite of sodium, unless they are quite faint and of a dull appearance. Furthermore, if any fluid containing both arsenic and antimony be introduced into the apparatus, the spots on the porcelain at first contain, principally, only arsenic, apparently in consequence of the antimony being less volatile; but if shining spots be produced upon the porcelain, which contain more antimony, these resist, more or less, the action of the hypochlorite of sodium, and are often eaten away only around the edges. While, therefore, by this reagent, a slight trace of antimony cannot be distinguished in spots of arsenical nature, arsenic, on the other hand, can be detected by it in antimonial stains.[14] The crusts, resulting from the presence of imperfectly charred organic matter in the suspected liquid, are not so readily soluble in nitric acid as are the arsenical crusts, and do not, like the latter, when so dissolved, yield a brownish-red precipitate with nitrate of silver.

Seventh. The following differential test is recommended by Taylor[15] as very reliable: "Receive the deposit from the burning gas on the interior of a small, white porcelain capsule. Add a few drops of strong nitric acid. The deposit will be immediately dissolved. Evaporate gently to dryness. Moisten the dry residue with one or two drops of water, and then add a few drops of a strong solution

[12] Month. Jour. Jan. 1853.
[13] Pereira.
[14] Wackenroder, Chem. Gaz. Aug. 2, 1852. This test was in use by Bunsen in 1844.
[15] On Poisons, Am. ed. 1875. p. 313.

of nitrate of silver. If the stain was owing to arsenic, wholly or in part, a brick-red colored precipitate will immediately appear. This will be more or less distinct, according to the quantity of arsenic present. The red precipitate (if owing to arsenic) is entirely soluble in ammonia. A deposit of antimony, thus treated, leaves a white residue (oxid of antimony), insoluble in water. Nitrate of silver added to it produces no colored precipitate; but if a little ammonia be brought near, either in vapor or liquid, and a solution of potash be added, a precipitate is formed, which becomes black by standing. Sulphid of ammonium dissolves the antimonial deposit immediately, and, on evaporation, leaves an orange-reddish colored film of sulphid of antimony soluble in hydrochloric acid, and insoluble in ammonia. The sulphid of ammonium does not readily dissolve the arsenical deposit; but when gently evaporated, it leaves a bright yellow film (sulphid of arsenic), not soluble in hydrochloric acid, but soluble in ammonia. Imponderable quantities of the two metals may be thus easily identified. In testing these minute films of arsenic, hydrochloric acid must not be used with the nitric, since, on evaporation, a portion or the whole of the arsenic may be volatilized, and lost as chlorid of arsenic."

Zinc, sulphuric, and hydrochloric acids are often contaminated by arsenic. The best answer to all objections, based on the presence of arsenic from accidental sources, is that the materials employed in analysis were tested repeatedly before the suspected liquids were introduced into the apparatus.

"If, in any case, we have no other evidence to offer than that furnished by Marsh's process,—a case in which the poison must be in infinitesimal quantity, and the metallic deposit proportionately minute,—it would be better to abandon the evidence altogether than to maintain the presence of poison from the results, which admit of no sort of corroboration; for all who have experimented upon the subject must have perceived the utter inefficacy of applying liquid tests to determine the chemical properties of imponderable and scarcely visible sublimates."[16]

176. Reinsch's test.— This method of separating arsenic is exceedingly simple and efficacious. A solution, supposed to contain arsenic, should be acidulated with hydrochloric acid, and heated to the boiling point. A thin leaf of copper, or fine, copper gauze or wire, bright and clean, should then be introduced, and if arsenic exists in the liquid, it will be deposited as an iron-gray film of the metal upon the copper.

[16]Taylor, op. cit. p. 316.

The copper, being removed after the deposit is formed, must be washed in distilled water, dried, and introduced into a reduction tube. On the slow application of heat, arsenious acid will be sublimed and deposited on the sides of the tube in the form of minute octahedral crystals. If the solution contained a very large amount of arsenic, so much metallic arsenic may be deposited upon the copper that, when heated in the reduction tube, the lower portion of the sublimate may consist of an arsenical mirror, and the upper portion, of the arsenious acid crystals. These crystals may be examined by a lens, and then dissolved in water and subjected to liquid tests. These supplementary tests are requisite, since solutions of various metals give a coating not unlike that of metallic arsenic, and if the copper is put into the acidulated fluid before it is duly heated, a stain will almost always occur in the presence of organic matters. In proof of the delicacy of this test, Prof. Rainey, of Glasgow, says that "in repeated experiments" he has "found that 1/1000 of a grain of arsenious acid in 1,000,000 times its weight of fluid, could be separated as a distinct deposit on copper.[17] The copper thus coated, when heated gently in a small tube, yielded a slight but distinct sublimate, most obvious on a black ground, and which, with a magnifying power of ten to twenty diameters, was found to consist of crystals with triangular facets, and which, when dissolved in water, yielded orpiment and the red arseniate of silver, when treated with the appropriate reagents." Dr. James St. Clair Gray read a paper to the chemical section of the Glasgow Philosophical Society, "On Certain Fallacies in the Means of Detecting Some Poisons," in which he pointed out the fact that Reinsch's test for arsenic is liable to fail, when the arsenic has undergone oxidation to arsenic acid, or when it exists in the state of sulphid. He advises the reduction of the arsenic acid by means of sulphite of an alkali; and in the case of the sulphid he would boil with caustic potash and dialyze.[18] The principal fallacy, connected with this test, exists in the fact that antimony may give precisely the same result, although usually the sublimate produced by heating the copper, upon which is a coating of metallic antimony, consists of prismatic crystals instead of octahedral; but the antimonial sublimate is very rarely in the form of octahedral crystals, so that this test is of little or no value in court as a means of distinguishing between arsenic and antimony. Reinsch's test is one of great

[17] $\frac{1}{50000}$ of a grain in the presence of 5,000,000 parts of liquid yields, after several minutes, a very distinct stain, the outer part of which has a dark, metallic appearance, and the inner, a brownish color. (Wormley, Micro-chemistry of Poisons, p. 287.)

[18] Brit. Med. Jour. March 16, 1872.

value as a preliminary test in legal cases, because the presence of organic matter does not interfere with it.

177. Bloxam's test.— Professor Bloxam has applied the process of electrolysis to the detection of arsenic. The apparatus proposed by him consists of a two or three-ounce bottle, the bottom of which has been cut off, and replaced by a piece of vegetable parchment, bound on by platinum wire. To the mouth of the bottle is fitted a cork with a bent tube, and a piece of platinum wire, which passes through the cork and turns up beneath in the form of a hook. A slip of platinum then hooks into the end of the wire, and passes nearly to the bottom of the bottle; it forms the negative pole of the arrangement. The bottle stands in an ordinary test glass, and the positive pole, also of platinum, stands in the glass. Dilute sulphuric acid is put into the bottle, and also in the glass, so as to stand at the same height in both vessels. The substance to be tested is introduced into the bottle, the cork adjusted, and the wires connected by five cells of Grove's battery; the heat of a spirit lamp is applied to the bent tube, and in the course of a quarter of an hour a distinct mirror is obtained, if arsenic is present. Standard solutions, containing respectively 1/10, 1/100, and 1/1000 of a grain of arsenious acid, were prepared and examined by this process, and in every case a successful result was obtained.

The solutions were mixed with organic substances, such as the ordinary articles of food,—meat, eggs, milk, etc.,—and the resulting matter examined. This was then dissolved by means of chlorate of potassium and hydrochloric acid, and the resulting fluid evaporated on a water bath, to a thick, syrupy fluid. The arsenic was thus obtained in a state of arsenic acid, which does not give a certain result by the electrolytic process. Some sulphurous acid was therefore added, and the mixture introduced into the bottle, after expelling the excess of sulphurous acid by evaporation; a dram of alcohol was then poured over the surface, and the process put into operation. . . . In all these experiments, of which a great number were made, 1/1000 of a grain of arsenious acid was readily detected.

The other metals, which may be detected by this process, are mercury, antimony, copper, and bismuth; lead is precluded by the sulphuric acid which is present. These are all precipitated in the metallic form upon a slip of platinum, and, even in the case of antimony, a mere trace of antimoniureted hydrogen is formed, the metal being deposited on the negative pole.[19]

[19] Brit. and For. Med.-Chir. Rev. April, 1860, p. 527.

178. Isolation of arsenic from organic mixtures.— Before the contents of the stomach, liver, spleen, or other organs containing arsenic by means of absorption during life, can be submitted to any of the tests for arsenic, it is necessary to obtain a solution as free as possible from organic matter. Various means have been recommended for this purpose; those which are the least open to objection are the following:

If it be intended to separate metallic arsenic by means of Reinsch's process, all the soft solids should be cut into small fragments, distilled water, if necessary, added, and also hydrochloric acid in slight excess. This mixture should be boiled gently for an hour, until all soft solids are either dissolved or broken down into fine flakes or grains. Filter through wet muslin, heat the filtered liquid again to the boiling point, and then introduce a slip of copper as before described.

If, however, the apparatus of Marsh is to be used, the following process is recommended by MM. Danger and Flandin: Add to the organic matter contained in a porcelain capsule one sixth of its weight of sulphuric acid, and heat until vapors of sulphurous acid appear. The matter is first dissolved, but during the concentration it is charred. The liquor is to be constantly stirred with a glass rod. The carbonization is effected without any swelling or frothing, and is to be continued until the charcoal is friable and almost dry. A small quantity of nitric or of nitro-muriatic acid is to be added by means of a pipette, when the capsule is cold. This converts the arsenious acid into the more soluble arsenic acid. The mixture is then to be evaporated to dryness, treated with boiling water, and the limpid liquor introduced into Marsh's apparatus, in which it never froths.

The best process, and the one most generally used in this country, not only for arsenic, but also for the other metallic poisons, is what is known as Flandin and Danger's process. It consists in finely dividing, with scissors, the solid parts, mixing them with portions of the liquids, adding a large excess of hydrochloric acid in an evaporating dish, and about a teaspoonful of crystals of chlorate of potassium. This mixture should then be heated over a water bath, when the solid portions will gradually become destroyed, and the fluid will assume a light yellow or green color; distilled water should be added to the mixture from time to time as the fluid evaporates and fresh crystals of chlorate of potassium, whenever the fluid becomes dark brown or black instead of remaining yellow. This should be repeated from time to time until the fluid remains light-colored after being heated at least half an hour after the last addition of potassium

chlorate. By this means the arsenic, if present, is converted to the form of arsenic acid. The mixture may then be allowed to cool, so that the fatty matters will separate, and filtered, the solids remaining upon the filter paper being thoroughly washed. The filtrate and wash-water united must be again heated over the water bath, until there is no more free chlorin remaining in the fluid. It is then necessary to reconvert the arsenic acid into arsenious acid, which is done by adding a saturated solution of sulphurous acid or of acid sulphite of sodium to the fluid, and continuing the heating over the water bath, until the odor of sulphurous acid gas has disappeared. Filter the fluid, if necessary, into a beaker or tall jar, and pass through it a slow current of sulphureted hydrogen gas for twenty-four hours. This will precipitate whatever arsenic is present, in the form of the yellow sulphid of arsenic; but it also precipitates some of the organic substances in the form of unknown organic sulphur compounds, which, unless arsenic is present in very large amounts, disguise the yellow color of the sulphid of arsenic; as a rule, the precipitate thus obtained has a dirty brown color. This precipitate should be separated from the fluid by filtration, and washed; if it contains sulphid of arsenic, the only impurity consists of a certain amount of organic sulphids, and must, of course, be further purified. This is done by treating the precipitate, while on the filter paper, with a boiling mixture of ammonia and ammonium sulphid, which will dissolve the sulphid of arsenic together with the organic sulphids; it will also dissolve any sulphid of antimony or sulphid of tin which may be present, but it will leave upon the filter paper any sulphid of lead, mercury, bismuth, copper, or cadmium, if present. The fluid passing through the filter should be collected in an evaporating dish and evaporated to dryness; usually the residue will have a dark brown or blackish appearance. The organic sulphids can now be destroyed by moistening with concentrated nitric acid, and evaporating, repeating this process three or four times, by which process also the sulphid of arsenic is converted into arsenic acid; or, by moistening with hydrochloric acid, adding one or two crystals of potassium chlorate, and evaporating to dryness. The arsenic is now left in the residue in the form of arsenic acid, which can be dissolved in hot, distilled water, and a portion of the solution subjected to all of the tests for arsenic acid (see below); another portion can be transformed to arsenious acid by acidulating with hydrochloric acid, and boiling with a solution of the acid sulphite of sodium until the odor of sulphurous acid has disappeared; this solution can be used for all of the tests for arsenious acid (see

above). All of the chemicals and pieces of apparatus used should have been tested and found to be absolutely free from arsenic, by having been employed in a previous or parallel analysis of tissues which contained no arsenic.

179. Arsenic acid, in general.— Arsenic acid is seldom met with outside of a chemical laboratory. It has been proved by experiments upon animals to be poisonous, and although it has given rise to no fatal cases of poisoning in man, yet its chemical study is of very great importance in legal cases, since, in the separation of arsenic from organic tissues, the white arsenic is always converted into arsenic acid. Some of the compounds of arsenic acid, which are formed by the tests used, should always be taken to court in legal cases as *corpora delicti.*

180. Tests for arsenic acid.— The principal tests are the following:—

(*a*) Ammonio-nitrate of silver.—This test is performed in precisely the same manner as for arsenious acid. The resulting arseniate of silver has a reddish-brown color, and is soluble both in an excess of ammonia and in free acids.

(*b*) Ammonio-sulphate of copper.—This test is also performed in the same manner as for arsenious acid, the resulting precipitate, arseniate of copper, having a bluish-green color instead of the light green, and is soluble in an excess of ammonia and free acids.

(*c*) Sulphate of magnesium.—If to a solution of arsenic acid be added a solution of chlorid of ammonium, a little ammonia, and a solution of sulphate of magnesium, and the mixture be shaken violently, or stirred with a glass rod, a white crystalline precipitate of ammonio-magnesium arseniate will be formed. This precipitate occurs in the form of prismatic crystals if slowly formed; but, in the form of fern-leaf crystals if rapidly formed. These crystals are exactly like those of the ammonio-magnesium phosphate, but can readily be distinguished from the latter by the fact that they yield metallic arsenic, when treated by the reduction test, Reinsch's test, or Marsh's test; and also yield a yellow precipitate, when dissolved in hydrochloric acid and treated with sulphureted hydrogen. This precipitate is also soluble in acetic acid.

(*d*) Nitrate of uranium.—A solution of arsenic acid is precipitated by a solution of nitrate or acetate of uranium, in the form of a pale yellow, flocculent or granular precipitate, which is readily soluble in free, mineral acids; therefore, it is always best to add a little acetate of sodium before adding the uranium solution, in order to neutralize any free, mineral acid which may be present. A similar

precipitate is formed by phosphoric acid, but can be distinguished in the same way as the ammonio-magnesium compounds; and, moreover, in the regular process of analysis in legal cases, the absence of phosphoric acid would be insured by the previous precipitation by sulphureted hydrogen and thorough washing of the precipitated sulphid. This test is especially valuable, because it can be used as a means for estimating the amount of arsenic quantitatively, as it is used for estimating the amount of phosphoric acid quantitatively.

Arsenic acid is also, like arsenious acid, precipitated by sulphureted hydrogen, although more slowly, because the sulphureted hydrogen must first convert the arsenic acid to the form of arsenious acid, which conversion is attended with the separation of sulphur, so that the precipitate formed consists of a mixture of sulphid of arsenic and sulphur. Arsenic acid also responds to Marsh's, Reinsch's, and the reduction tests like arsenious acid.

181. Sulphids of arsenic, in general.—There are several of these compounds known in commerce; as realgar, of an orange-red color; orpiment, which is yellow; and (it is said) another preparation, bearing the same name, which is a compound of pure sulphid and arsenious acid. The pigment, known as king's yellow, contains a sulphid of arsenic and a considerable proportion of lime and sulphids. Cases of intentional and accidental poisoning with orpiment are known. A female was poisoned with it in England in 1835, and the poison was found in considerable quantity in the stomach of the deceased fourteen months after death. Its character was satisfactorily proved by chemical analysis, and led to the apprehension and conviction of the murderess, who was afterwards executed.[20] Another case, in which it was mixed in porridge, by mistake for turmeric, is related by Dr. Jochner. An old man and his nephew both partook of the food, without immediately discovering the mistake. The prominent symptoms were continual vomiting, burning pain in the stomach, and gradual collapse. The old man died in twenty-two hours; the boy escaped. Evidence of violent inflammation was found in the œsophagus and stomach, the mucous coat of the latter being soft and thickened. There was a sphacelated spot, 1 inch in diameter, in the œsophagus, and another in the stomach of 3 inches in extent.[21]

The sulphids of arsenic, when pure, are not poisonous; but when exposed to a moist atmosphere, they become partially oxidized, and

[20] See the case quoted in Beck, Vol. II. p. 560.

[21] Henke's Zeitsch. Erg. II. 43, 162.

thereby mixed with arsenious acid. All of the cases of poisoning reported are, in reality, cases of poisoning by arsenious acid.

These sulphids, when pure, are insoluble in water or hydrochloric acid, but are soluble in hot, concentrated nitric acid, being converted into arsenic acid, and are also oxidized into arsenic acid by hydrochloric acid and potassium chlorate. They yield metallic arsenic, when subjected to the reduction test. They are soluble in the alkalies. When impure, that is, when they contain, as they usually do, more or less arsenious acid, they of course respond to all of the tests for arsenious acid.

182. Arsenite of potassium, in general.—These and other compounds of arsenious acid with alkaline bases are poisonous. The solution of arsenite of potassium is of much use in medicine,—especially in the treatment of chronic skin diseases and intermittent fever,—under the name of Fowler's solution or liquor potasii arsenitis (U. S. P.). It receives its taste and color from the spirit of lavender, as officinally prepared, and contains 4 grains of arsenious acid to the fluidounce, the usual dose of which is, for an adult, 5 to 15 minims three times a day. From the occasional adulteration of arsenic with the sulphate of lime, this preparation is no doubt sometimes weaker than the officinal strength. Two cases of fatal poisoning by it have been before referred to. In one of them the dose did not amount to more than 2 grains in five days. In the other the amount swallowed was not ascertained. The symptoms are the same as those produced by arsenious acid. The dose, however, which will produce poisonous effects is very uncertain. This liquid may be tested, after acidulation with hydrochloric acid, by means of any of the tests for arsenious acid in solution.

There are a number of solutions of arsenious acid in alkalies used for various purposes; thus, solutions of arsenious acid in soft soap are sometimes used for washing sheep, and have given rise to cases of poisoning. Taylor records cases of poisoning due to arsenite of sodium, which was used as a cleansing preparation; there is a soap (called "savon de Bécœur") employed by taxidermists for the preparation of skins, which contains 32 per cent of arsenious acid.

183. Arsenite of copper, in general.—As this article owes its poisonous properties rather to the arsenic contained in it than to the oxid of copper, we have ranged it with the arsenical compounds. It has frequently occasioned accidents in England and on the continent from its use as a coloring ingredient in confectionery. In 1850, Dr. Letheby reported three cases of poisoning by Scheele's green, which

came under his notice; and he states that between thirty and forty children were poisoned at the same time by sweetmeats sold to them by a confectioner in Petticoat Lane. He stated, moreover, that as many as seventy cases of poisoning had been traced to a similar source within three years.[22]

Within the past few years, in this country, there has occurred a very large number of cases of acute poisoning, mostly suicidal, from another compound, which can always be bought by the pound at almost every country store, for the purpose of killing potato bugs; it is sold under the name of Paris green. The writer has seen four or five cases within the last two years. The symptoms caused by large doses of Paris green are exactly the same as those caused by arsenious acid, with the exception of the coppery taste and the green color of the vomitus and stools. The symptoms caused by this substance, when slowly absorbed, as in cases where it is used for coloring wall paper, have already been mentioned.

The arsenite of copper, when pure, can be very easily recognized, since it responds to the tests for both arsenic and copper. Its bright green color will usually lead at once to a suspicion as to its nature. A small portion of the powder, treated with a drop of ammonia upon a watch glass, immediately imparts to the latter an azure-blue color; and if a drop of a solution of nitrate of silver be allowed to flow down the side of the watch glass, so as to form a separate layer of fluid under the ammoniacal solution, a lemon-yellow precipitate of arsenite of silver will be seen at the junction of the two fluids. This test will serve to detect the pigment in wall paper, when no other pigments are present. If a little of the powder be heated in a reduction tube, it will be decomposed, arsenious acid will be volatilized and condensed on the cool portion of the tube, and oxid of copper will remain at the bottom. These tests will serve to detect the nature of Paris green quickly; but, in legal cases, they must be confirmed by applying all of the tests both for arsenic and for copper.

In order to isolate this compound from organic mixtures, the same process should be resorted to, which has already been described in detail under arsenious acid (*supra*, § 178).

XII. Phosphorus.

184. Different forms and modes of administration.— Phosphorus, as

[22] Brit. and For. Med.-Chir. Rev. July, 1851.

a poison, was almost unknown previous to the year 1850, when its poisonous properties began to be understood by the laity; it gave rise to many cases of poisoning, since it could be so easily procured. The cases of phosphorus poisoning in this country are chiefly accidental, due to the ingestion of the phosphorus ends of matches by children; some cases are caused by the taking of vermin pastes which contain phosphorus, and which are usually mixtures of meal or flour with some coloring matter, like Prussian blue or Venetian red, and some flavoring matter suited to the taste of the animal for which they are intended. Commercial phosphorus exists in two forms, the transparent or common form, which is poisonous, and the amorphous or red phosphorus, which can be commonly ingested, when pure, with impunity. The form, which is usually employed in the manufacture of matches and vermin or animal pastes, is the transparent or poisonous variety. In the manufacture of matches but little phosphorus is required, a pound of phosphorus sufficing for nearly one half million of matches. Matches are usually first dipped in an emulsion of sulphur and glue, or some other glutinous material, and then the ends tipped with a mixture of phosphorus, coloring matter, as vermilion, oxidizing material like potassium chlorate, and glue or mucilage. In the so-called safety matches, all of the phosphorus is placed upon the box or scratching surface. Matches are the most frequent source of phosphorus poisoning, and have been the cause of criminal, accidental, and suicidal poisoning; most of the cases are accidental, caused by the sucking of the phosphorus end of matches by children. In criminal cases, matches are soaked in some article of drink like coffee or tea: but the mistake is often made of not soaking the ends sufficiently long, or of not stirring the fluid, in both of which cases, all, or nearly all, of the phosphorus remains at the bottom of the cup or other vessel: or, of soaking the ends too long, in which case the phosphorus is oxidized and transformed into a comparatively harmless compound, phosphorous or phosphoric acid.

Phosphorus exists in four forms, two of which are very rare and hardly known outside of a chemical laboratory, and the remaining two, one, the yellow, waxy form, the other, the red variety, are common articles of commerce.

The waxy or crystalline form is generally sold in translucent, cylindrical sticks, has the consistency of wax, and, as it oxidizes rapidly, is best kept under water. It ignites at 50°C; hence great care must be exercised in handling it, as it produces severe burns if it catches fire. It has a garlicky odor, is very slightly soluble in water, more so in

alcohol, and quite so in carbon disulphid, ether, etc. It emits a feeble light, and when exposed to the air, fumes of its oxid are given off.

Red phosphorus, an allotropic form, is obtained by heating the waxy variety to 240° or 280°C. in an atmosphere of carbon dioxid, for some time, and washing the product with carbon disulphid. Prepared in this way, however, it is rarely free from traces of the yellow variety. Pure, red phosphorus is nonpoisonous. It is a reddish-brown, amorphous body, odorless, tasteless, and ignites at a much higher temperature than the yellow form, about 250°C.

The ordinary sulphur match is tipped with yellow phosphorus, sand, glue, and potassium chlorate. The so-called safety match generally has sulphid of antimony in place of the phosphorus, and red phosphorus is put upon the box or scratching surface.

There is one other compound of phosphorus which is very poisonous, and had better be mentioned here, *viz.*, phosphine or phosphureted hydrogen. It is found, when nascent hydrogen comes in contact with phosphorus or its lower oxids. It is colorless, sparingly soluble, and ordinarily inflames on contact with air. If formed with the evolution of hydrogen, it burns with a greenish flame, and shows three characteristic absorption bands in the green, with the spectroscope.

185. Preparations.— (U. S. P.) Phosphoric acid; diluted phosphoric acid containing 10 per cent by weight of absolute ortho-phosphoric acid; elixir of phosphorus, each cubic centimeter representing ¼ of a milligram (0.00025 gm.) of phosphorus; phosphorated oil containing 1 per cent of phosphorus; spirit of phosphorus containing 1.2 grams in 1000 cubic centimeters of absolute alcohol; pills of phosphorus containing 6 centigrams (0.06 gm.) in each 100 pills.

In Mr. Blyth's table for ten years ending 1892, there are 92 reported deaths by suicide from phosphorus, and 55 from accident or negligence. Of this latter class, 26 children under five are included, and 3 between the ages of five and fifteen. Under the head of suicide, 7 of the deaths were from five to fifteen years of age. Mr. Blyth calls attention to the fact that there are more cases of fatal poisoning from phosphorus in France and Germany than in England; and that, in a series of 108 cases of poisoning collected by Meischner, together with 16 coming under the observation of Schraube, only 18 persons recovered, and 90 died. These 108 cases occurred between the years 1798 and 1868. Of these 71 were suicidal, 21 were those of murder, 11 were accidental, and the history of the other five was not known. Falck also collected, in a later eleven years, 76 cases of phosphorus poisoning from various sources. Of these 55 were suicidal and 5

homicidal, the remainder accidental. The mortality in Falck cases was 55.3 per cent. Witthaus and Becker, out of a set of 522 cases, determined that in only 10 was the substance other than that of matches or vermin poison. Of these 522 cases, 110 were homicidal, 294 suicidal, 66 accidental, 4 abortion, 1 experiment, 2 quack. Only 3 of the homicidal and 3 of the suicidal cases occurred in the United States. In the Massachusetts medical examiners' returns for twenty-five years, there were 17 deaths caused by poisoning from phosphorus; 11 of these were suicidal, and 6 were accidental; 2 of the latter were from matches sucked by children, 1 from vermin exterminator.

186. Symptoms of poisoning.— Phosphorus is one of the most insidious poisons with which we have to deal, since the symptoms may be quite slow in making their appearance, and in different cases may assume an entirely different character, so that we may have three distinct kinds of symptoms in phosphorus poisoning, according as they are chiefly irritant, as in the usual form of poisoning, or chiefly affect the nervous system or the blood, although in some cases we see symptoms belonging to all of these different varieties. In all cases there is usually an immediate eructation of gas which has the taste of phosphorus, and, if the mouth and throat be viewed at this time in the dark, luminosity will be perceived. The severe symptoms do not usually appear for several hours, generally in five or six hours, but sometimes earlier, and sometimes not for eighteen or twenty-four hours. Usually, after the lapse of several hours, there is pain in the throat and a sensation of heat in the epigastric region; the tongue is swollen; there is a general feeling of malaise, nausea, and vomiting; the vomitus may be bloody, but usually is not, and if it contain free phosphorus, as it generally does, it will be luminous in the dark,—especially after being shaken with air; then colicky pains and diarrhea come on, with tenderness of the abdomen. These symptoms last for one or two days, when they may cease entirely, although the pulse remains feeble, and pain still exists in the limbs and back; and, as the patient is, apparently, convalescing, death may take place suddenly in from two to four days. In many cases, between the second and fourth day, the patient becomes jaundiced; there are headache, insomnia, retention of urine, which is frequently albuminous, owing to the affection of the kidneys caused by the phosphorus, and contains the abnormal sediment characteristic of the affection; then vomiting takes place at intervals; the stools are frequent, painful, and sometimes involuntary; finally, delirium comes on, followed by coma and

death, in from about six to twelve days. These symptoms, which are the most common, resemble somewhat those of so-called, acute, yellow atrophy of the liver, although some authors doubt the existence of such a disease. In some cases in which the effects of the poison upon the nervous system predominate, the symptoms begin at about the same time; there is the same soreness of the throat, and pain in the epigastrium, with nausea, which may or may not be attended with vomiting; then, there may appear sensations of numbness or tingling in the limbs, with cramps and repeated fainting, and there is very great prostration; an erythematous eruption may appear, and, on or about the fifth or sixth day, delirium comes on, with the uttering of loud cries, gnashing of teeth, convulsive twitchings, coma, and death in from six to twelve days. In still rarer cases, the phosphorus affects the blood, destroying the blood globules and setting the pigment free, so that we see the evidences of this action. In these cases the progress of the case is generally slower, although the onset of the symptoms is the same, and the vomitus is more apt to be bloody. There are also bloody stools, with tenesmus; the liver becomes gradually enlarged, and there is very great weakness and prostration. After a few days amelioration takes place, although colic occurs occasionally, and in a few weeks hemorrhages begin to occur from all of the mucous membranes of the stomach, mouth, throat, nose, uterus, ears, etc., etc.; finally, the blood becomes fluid, and extravasations take place beneath the skin and mucous membranes; dyspnea cardialgia, and general numbness occur from deterioration of the blood; death may take place after the lapse of perhaps several months, from exhaustion caused by the numerous hemorrhages.

Cases of professional poisoning have occurred, but are prevented by exercising due care in the selection of employees. It has been noticed that those who have sound teeth are not affected with the disease, which consists of a necrosis of the jaw, so that it is now a practice, in manufactories of phosphorus matches, to employ only those who have sound teeth.

187. Forms of poisoning in general.—It would be difficult to treat poisoning from phosphorus in the way that we have described the effects of other poisons; *viz.*, by describing the three forms of poisoning as acute, subacute, and chronic. Tardieu's careful studies of the effects of phosphorus poisoning have, for a long time, been the guide for toxicologists for the discussion of the symptoms following the ingestion of this poison. This writer, from a careful research of clinical notes of a number of cases under observation, divides the effects

of this poisoning under the head of "common form, hemorrhagic form, and nervous form." He remarks:[23] "I think it useful to establish that poisoning by phosphorus, in its course sometimes rapid, sometimes slow, exhibits in its symptoms three distinct forms,—a common form, a nervous form, and a hemorrhagic form. I recognize that in certain cases these three forms may succeed each other, and may only constitute periods of poisoning; but it is incontestable that each of these may show itself alone, and occupy the whole course of the illness produced by the poisoning." In commenting upon this classification of Prof. Tardieu, Mr. Blyth gives the result of his study of 121 recorded cases of phosphorus poisoning: In the common form, 83 per cent of these cases were classed, in the hemorrhagic form, 10 per cent, in the nervous form 6 per cent, unclassified 1 per cent.

188. Common form.—Immediately after swallowing the phosphorus, there is a disagreeable taste and odor experienced. Immediately following this, an intense pain and depression in the region of the stomach is felt, increased by pressure over the pit of the stomach; eructations, which occasionally appear luminous in the dark; this may continue for a period of three or four days. Immediately following the distress in the stomach, nausea and vomiting take place; the throat and tongue become swollen and painful. These symptoms do not always appear immediately, but may be delayed from one to six hours, the phosphorescent eructations appearing before the distressed symptoms. The vomited matters are often phosphoric, and tinged with blood. Diarrhea occurs, although it is not always prominent. There is also restiveness and sleeplessness for the first few nights. The pulse may be slow, in some instances, and hastened; the bodily temperature is usually lower than normal in the mornings, and normal in the evening.

Jaundice appears, in a few cases, within two or three days, but is more usual within three days, and may even be delayed until the fifth day; occasionally, it may delay from two to four weeks before its appearance. In Mr. Blyth's cases, jaundice appeared before the end of the third day in 78 per cent; and in 26 of these cases, in which the patients lived long enough for the occurrence of jaundice, this symptom was absent in 3. In 132 cases recorded by Lewin, Meisehner, and Heisler, jaundice appeared in 49 per cent; but in many of these cases the victim died before jaundice had time to develop. Where

[23] Etude Medico-Légale et Clinique sur l'Empoisonnement, Paris, 1875, p. 483.

jaundice is a pronounced symptom, the usual incidents peculiar to jaundice become prominent; in fact, these may be similar to those accompanying acute, yellow atrophy, such as retention of urine, sleeplessness, headache, frequent vomiting, painful and involuntary intestinal excretions, sometimes itching eruptions on the skin. When jaundice has occurred as a prominent symptom, following the enlargement of the liver, recovery is unusual; but when recovery occurs, it is usually preceded by a diminution in the size of the liver. Jaundice, which we may view as a traumatic lesion of the liver, or intoxication, may be considered as the most dangerous symptom, in phosphorus poisoning, of a fatal issue.

189. Hemorrhagic form.— The symptoms of this form may originate with those described in the preceding paragraph; but, in the present class, there is the additional, periodic symptom of a tendency to general hemorrhage, bleeding from the mouth, nose, and from the passages lined with mucous membrane. This hemorrhage may also occur in a subcutaneous tissue, in the form of purpura, ecchymoses; the skin and mucous surfaces may appear mottled from this extravasation of blood in the subjacent tissues. Great prostration accompanies this stage, and this condition may last for a long period of time, the victims dying weeks or months after having absorbed phosphorus, and after these suffering symptoms of its intoxication. The urine is scanty, high-colored, containing albumin, bile pigments and casts; hemoglobinuria.

190. Nervous form.— This form, in which derangements connected with the nervous system are the predominating features, is not so common as the two preceding forms of phosphorus intoxication. The first symptoms observed in this class are strange, creeping sensations in the muscles of locomotion, spasmodic cramps, fainting spells, and intense sleepiness. Jaundice, purpura, appear about the fifth day, followed by an acute delirium, lockjaw, convulsions, and death.

191. Lethal dose.—A young man died on the twelfth day, from the effects of 1½ grains (0.097 gm.).[24] Martin-Solon relates the case of a patient who died in two days from less than a grain in the form of emulsion.[25] An apothecary named Doffenbach, in experimenting upon the effects of this substance, took 1 grain (0.65 gm.), the next day 2, and the following day 3 grains; three days after the last dose, he was seized with violent vomiting, and died on the seventh day.[26] A child two and a half years old died after swallowing the phos-

[24] Worbe, Med.-Chir. Zeit. 1826, Bd. 4, p. 183.

[25] Christison, p. 151.

[26] Froriep. Notiz. Nr. 493.

phorus on eight friction matches.[27] A child two months old is said to have died from the effects of two such matches.[28] If of the average quality, they would contain about $\frac{1}{50}$ of a grain of phosphorus.

The smallest fatal dose recorded for an adult is .116 of a grain (7.5 mg.); for an infant five weeks old, $\frac{1}{50}$ of a grain (0.13mg.) of phosphorus contained in a single match head. It should be noted that phosphorus is exceedingly poisonous, when it enters the body in minute portions, or in the form of solutions; but if taken in coarse pieces, it may not then be absorbed, but passed out undigested through the whole length of the intestinal canal. Majendie introduced phosphorated oil into the venous circulation of animals. They exhaled white fumes, some few dying from asphyxia; but no symptoms of phosphorus poisoning were observed. These experiments, repeated and confirmed by others, seem to prove that these coarse particles of phosphorus were arrested in the capillary vessels of the lungs, which interfered with pulmonary respiration. According to experiments, performed under the direction of L. Hermann, consisting of the injection of a fine emulsion of phosphorus into the venous system,[29] the phosphorus in its divided form passed through the pulmonary capillary blood vessels, in this case, without the animals exhaling white fumes, and, being absorbed, soon manifested the symptoms of phosphorus poisoning. Therefore, as phosphorus paste and the heads of matches contain phosphorus in a minutely divided form, the ingestion of this form of phosphorus is, on account of its easy absorption, extremely poisonous.

Mr. Blyth concludes, from his study of the clinical history of fatal cases of poisoning by phosphorus, that, though 6 of these cases showed symptoms commencing within the hour (about half of the cases), 80 per cent of these cases may be expected to show poisonous symptoms within six hours. The above writer finds that in 150 cases of fatal poisoning by phosphorus, 17 died within twenty-four hours, 30 within two days, and 103 within seven days. Three patients lived eight days, 6 nine days, 13 ten days, 1 eleven days, 1 sixteen days, 1 seventeen days, and 1 survived eight months. This showed that 79.8 per cent of these fatal cases died within one week.

192. Vapor of phosphorus.— The vapor of phosphorus, which we observed in a former paragraph has caused professional poisoning

[27] Schmidt's Jahrbücher, 1844, No. 6, Bd. XLII.

[28] Husemann, Jour. f. Pharm. II. 169.

[29] Arch. f. d. Ges. Physiologie, III. p. 1.

among employees in factories where phosphorus is used, has, in close, ill-ventilated apartments, subjected a workman to fatal poisoning from inhaling phosphorus vapor.[30] Another case is mentioned by Mr. Blyth, in which a man, thirty-nine years of age, was working in a match factory at Stockholm in 1842. By some accident the material for making these matches took fire and exploded. The inhalation of the phosphorus vapor caused this man to fall down unconscious; he could not get up, having lost, in a great measure, power over his legs and arms. He was taken to the hospital, where, after a little while, he could take a few, staggering steps, but was in a very paralyzed condition, with spasmodic twitching of his muscles, but with no pain. The sensibility of the skin and of the region over the spine was neither sensitive to pressure, nor to heat applied from a hot sponge; his speech was somewhat obscure; but the symptoms of paralysis gradually increased for three years, when he died. A peculiarity of the chronic, phosphorus poisoning, experienced in factories among their workmen, is illustrated by the following case reported by Fournier and Olliver,[31] and exhibits the sudden access of phosphorus vapor poisoning in a girl, after working four years in a phosphorus factory; the symptoms were periosteitis of the upper jaw and intense anemia. After the eruption of purpura, she died comatose. Wegner[32] found, after administering small daily doses of phosphorus to animals, both as vapor and as finely divided solid, their condition showed that the bones were more contracted than normal, the medullary canals being smaller than in healthy bones. Ossification was hastened, and the formation of callus in fractured limbs was increased.

193. Treatment.— The true antidote for poisoning by phosphorus, after emptying the stomach either by medicine or stomach pump, is oil of turpentine, in full medicinal doses of about 40 drops (2.5 c.c.), frequently administered. This method of treatment, when resorted to at an early period after the ingestion of the poison, has caused many recoveries. Sulphate of copper, as an emetic, should be avoided, as it may cause severe gastroenteritis.

194. Action.— According to the investigations of Dybkowsky,[33] it seems probable that the principal action of phosphorus is due to the formation of phosphureted hydrogen in the alimentary canal; this

[30] Annuaire de Thérap. 1874, p. 109; Schuchardt in Maschka's Handbuch; also Schmidt's Jahrbuch, 1846, Bd. 51, s. 101.

[31] Gaz. Hebd. de Méd. 29, p. 461, 1868.

[32] Virchow's Arch. f. Path. Anat. LV. II.

[33] Hoppe-Seyler's medicinisch-chemische Untersuchungen, 1866, I. p. 49.

can readily take place, since nascent hydrogen is produced during the natural processes of digestion. The local inflammatory effects can also be produced, when the poison is taken in tolerably large pieces; but many cases are reported,—especially those in which the phosphorus is taken in a finely divided state,—where there is no evidence of any local inflammatory or ulcerative action.

It should be observed that red phosphorus, made by heating phosphorus in a vessel closed without air for thirty-six hours, is a reddish-brown powder, which does not freely oxidize in air and requires a higher temperature for combustion than ordinary phosphorus, and is not poisonous when pure. The commercial article exists with a small percentage of stick phosphorus.

It would naturally appear, from the peculiarities noticed in some of the clinical cases of phosphorus poisoning, that this poison must produce some morphological alteration of the blood, when this substance is absorbed; but the authorities appear to differ as to the evidence that phosphorus prevents coagulation of the blood. Nasse maintains that phosphorated oil, given to a dog, prevents coagulation of the blood; on the other hand, Liebreck[34] found the blood dark, fluid, and "in perfect solution;" his observation was confirmed by Bibria and Schuchardt.[34a] Lebert and Wyss found the blood of animals, both in veins and inextravasations, in a normal condition. Phosphorus increases the fatty contents of the blood. Ritter observed that when phosphorus mixed with starch was given to a dog the fatty elements increased from 2 per 1000 up to 3.41 and 3.47 per 1000. Memard[35] observed that the blood of the jugular and portal veins, as well as in the extravasations, produced microscopic, fat globules, and hematin crystals soluble in ether.

It has been noted in the description of symptoms of poisoning by phosphorus that these indicate an alteration in the cell tissue of the liver and kidneys. It would appear, also, that almost all of the organs of the body show a degeneration of the tissue structure. It is quite evident that the action upon the liver tissue is very marked, not only on account of the subsequent appearance of jaundice, but also because leucin and tyrosin, products of the liver secretion, are passed out with the urine through the kidneys. The urea is much diminished, and may be, towards death, entirely absent. Kobert inclines

[34] Diss. de Venefico. Phosphoreo Acuto, Upsal, 1845.

[34a] V. Bibra u. Geist. Die Krankheiten der Arbeiter in dem Phosphorzundholz Fabriken, 1847, s. 59, &c.; Henle u. v. Pfeuffer's Zeitschr. f. ration. Med. N. F. Bd. 7 Hft. 3, 1857.

[35] Etude Expérimentale sur quelques lesions de l'Empoisonnement aigu par le Phosphore (Thèse), Strasburg, 1869.

to the opinion that there are grave disturbances of metabolism. Degeneration of the body albumin produces fat, leucin, tyrosin, sarcolactic acid, and phosphorus bases. He also notes that the kidneys are shrunken, and that the tissue of the heart undergoes degeneration.

195. Diagnosis.— To establish the diagnosis of poisoning by phosphorus, the evidence of its odor and phosphorescence in the exhalation from the lungs, and in the vomited material from the stomach, would determine the fact; subsequently, the purpura and ecchymoses under the skin, taken in connection with the jaundiced color of the skin, would, with the preceding symptoms, offer corroboration; finally, the actual chemical tests of the presence of phosphorus in the urine would insure diagnosis.

196. Post-mortem appearances.— It must be noted that the post-mortem appearances vary with the form of phosphorus used.

1st. When pure phosphorus has been used alone, or in combination with fat or oil, the œsophagus and digestive canal are more often the seat of pathological alterations. "Fragments of solid phosphorus, recognizable by the odor or by the phosphorescence, have been found adherent to the mucous membrane of the intestines, and even in the large intestine."[36] In the œsophagus and digestive canal may be found, here and there, ecchymotic or gangrenous spots. Generally the mesenteric ganglia are engorged, softened, and friable.

2d. When this poison has been in the form of paste, or the substance used on matches, it will often happen that no appreciable anatomical lesion can ever be detected; but ordinarily, if there be neither redness, trace of inflammation, nor ulceration, very many points of hemorrhage may be determined. On opening the abdomen, the mesentery and the visceral peritoneum appear spotted with black ecchymotic patches or points, analogous to the spots in purpura. The blood is liquid, without any notable alteration of the blood corpuscles. The bladder incloses urine mixed with blood, and presents, oftentimes, submucous ecchymoses.

The microscopical examination of the tissues is an important means of detecting poison by phosphorus. The liver, the kidneys, and the follicles of the stomach, the heart, and muscles are seen under the microscope in a condition of fatty degeneration. Still, these peculiarities *per se* are not indicative of poisoning by phosphorus, as they are found after poisoning by many other agents, and also as the result of many diseases of the lymphatic system. These appearances must be associated with some of the important symptoms of phosphorus poi-

[36] Tardieu, op. cit. p. 437.

soning, as the icteric appearance of the body, muscular pains, and weakness. Tardieu[37] gives a representation by plates of the microscopical appearances mentioned above.

In one case, the mucous membrane of the stomach was of a crimson color, softened in many places, and easily detached; near the pylorus was an ulceration of the size of a quarter dollar, with brown everted edges, and the muscular coat under it bare. Another similar ulceration was found in the greater curvature. The whole of the small intestine exhibited signs of violent inflammation, but the large intestine was free from it, except in the rectum. In another case, the mucous coat of the stomach and duodenum was so softened that the handle of a knife, passed behind it, readily detached it in a softened condition. Similar conditions have been found in still other cases.[38] These morbid alterations, therefore, resemble those of gastroenteritis, arising from other causes. The agency of phosphorus, in producing such changes, must be ascertained, not only from the history of the case, but also from its detection by the senses, and by chemical examination. Krahmer states that phosphorus, after absorption, so affects the blood as to produce ecchymoses under the mucous membranes, skin, etc.[39] It should, however, be borne in mind that fatal poisoning by phosphorus may take place without the stomach or, indeed, any of the organs displaying the slightest lesion. This is shown in Nitsche's case,[40] in which the dose of the poison was very large; in that quoted by Husemann;[41] in Lewinsky's case,[42] and others.

One of the most common alterations is a remarkable fluidity of the blood. Nitsche, Lewinsky, Krahmer, and Husemann describe it. Of this point, Casper remarks that we must admit phosphorus to be sometimes fatal by destroying the vitality of the blood. In the cases reported by him, the blood disks had become transparent by the loss of their coloring matter, which was diffused through the uncoagulated plasma, giving it the appearance of a cherry-red fluid of syrupy consistence. Such changes prove the blood to be deprived of vital and nutritive qualities.

The contents of the stomach or the matters vomited may give a white inflammable vapor, and be luminous in the dark. This was

[37] Op. cit. p. 441.

[38] In two cases, one of which was American, they were present in various degrees from light to dark injection, and thence to grayish and gangrenous ulceration. See Boston Med. and Surg. Jour. Nov. 1855, p. 323; and Nov. 1858, p. 343.

[39] Handbuch 2te Aufl. p. 462.

[40] Zeitschrift der k. k. Gesellschaft der Aertze zu Wien; and Am. Jour. Med. Sci. Jan. 1858, p. 290.

[41] Jour. f. Pharm. II. 169.

[42] Brit. and For. Med.-Chir. Rev. Oct. 1859, p. 529.

observed in Lewinsky's case, referred to above. They may also exhale a phosphoric odor. Flachsland reported a case in which the dejections obtained by enemata were luminous in the dark, and pieces of phosphorus were found in them.[43] It is said that the intestines, and even the flesh of animals poisoned by phosphorus, have the odor of garlic, and appear luminous in the dark. In a woman, who died while taking phosphorus medicinally, it was remarked that the whole of the viscera of the body were luminous; thus indicating the extensive diffusion of the poison.[44] Brera observed also, in opening the body of a woman to whom he had administered phosphorus both by the mouth and rectum, that a white vapor, having an alliaceous odor, and taking fire at the approach of a flame, arose from the stomach.[45] In the report of a case by Dr. Bingley, in 1857, it is said, "on opening the stomach there was an escape of white smoke, accompanied by a strong smell of garlic." [46] Similar vapors, luminous in the dark, have been seen issuing from the rectum and even from the vagina.[47] Another case is interesting from the fact that, although the body had been buried fourteen days, phosphorus was discovered, by means of its physical properties, in the stomach.[48] On the tenth day after it had been taken, Mayer detected it in the contents of the intestine.

The above cases prove that sometimes a portion of the phosphorus may be absorbed as such into the blood, and impart its luminosity to an internal organ; the same thing has been demonstrated by experiments upon animals, the breath exhaled being luminous, after the phosphorus had been introduced into the stomach through an external opening, after the œsophagus had been tied.

In reference to the confusion which might arise in differentiating the disease of acute, yellow atrophy of the liver from the yellow atrophy which occurs after poisoning by phosphorus, we would quote the words of Dr. Hans Schmaus:[49] "Acute, yellow atrophy is a rare disease which, at times, follows infectious and septic conditions, and may occur during pregnancy or with puerperal fever. It is more common in women than in men.

"A condition, similar to the above, follows phosphorus and arsenic poisoning. The essential element is fatty degeneration. Since death occurs earlier than with acute, yellow atrophy, often in a few seconds, and at the longest after a few days, the liver may be studied in an

[43] Med.-Chir. Zeit. 1826, IV. p. 183.
[44] Taylor on Poisons, p. 244.
[45] Riflissioni Med. Prac. sul'uso interno del fosforo, etc. Pavia, 1778, p. 8.
[46] Lancet, June, 1857, p. 600.
[47] Casper, Gericht. Med. I. 401, 442.
[48] Schaffer, quoted in Henke, Zeitschrift, 1851. E. II. 43, p. 215.
[49] Op. cit. p. 396.

early stage, when there is no removal of the degenerated tissues, but when excessive fatty changes are beginning the process. The liver is then large, icteric, soft, and friable. If death is delayed for a time the condition reproduces that of yellow atrophy.

"In all three conditions the heart, the kidneys, and the alimentary epithelium suffer albuminous and fatty degeneration. With the poisons mentioned the striated muscles present the same changes."

It should be noted that, where there are scattered spots of degeneration in the liver tissue, due to lesions effected through closure of small blood vessels in that organ, or from anemic or hemorrhagic infarction, this condition should not be confused with the universal, cell degeneration following poisoning from either phosphorus or arsenic. The same remark applies to the scattered areas of fatty degeneration which is met with in typhoid, recurrent fever, variola, scarlatina, malaria, Weil's disease, and puerperal eclampsia. It should also be noted that, with the degeneration of the liver tissue, there is coexistent,—especially after the lapse of a few days,—fatty degeneration of the heart tissue, kidneys, and voluntary muscles. As between the post-mortem effects from arsenical poisoning, they are distinguished from those of phosphorus, as apart from the phosphorescent odor, etc., mentioned above. There should be called to mind the effects of arsenic upon the question of the resistance which arsenic affords to the cadaver to putrefaction.

There are certain cases, mentioned above, wherein it is noted that the post-mortem appearances, after fatal poisoning, did not display any lesion; as justly remarked, in discussing the case quoted by Prof. Tardieu of this character, that the microscope was not used to examine, in these earlier cases reported, the microscopical character of the liver cells, and the tissues of the other organs in which the lesions usually occur; consequently, without the use of the microscope these specific lesions might go unnoticed. This condition of the degeneration in the liver and other organs has been very carefully considered, and illustrated by undoubted cases recorded by observers; and it is now generally admitted by writers on toxicology that this evidence of poisoning is one of the most constant, and appears at a very early period after the swallowing of the poison. The experiments of feeding phosphorus to animals reveal the same early appearance of the alteration in the structure of the organs above enumerated. Acute, yellow atrophy seems to be followed, when occurring as a disease, by an inflammation of the connective tissue between the lobules of the liver; while, in phosphorus poisoning, the cells are swollen and con-

tain fat globules, and, in this condition, press upon the vessels, thus shutting off the blood supply and further wasting of the tissue of the liver. Maschka emphasizes the fact that there are never found, in cases of phosphorus poisoning, the grey-white, knotty, fecal masses which are usually in the intestines during acute, yellow atrophy; it is more common to find, associated with phosphorus poisoning, the fecal masses fluid, and accompanied with catarrh.

197. Chemical examination.— The appearance of phosphorus is familiar to everyone. It is insoluble in water, but soluble in ether, alcohol, and the oils. It melts at 110°, and takes fire at a temperature a little above this. It has a peculiar odor, which may readily be perceived by rubbing the moistened finger over the phosphorus end of a match. In contact with air or oxygen it is luminous, if examined in a dark room. Sometimes it may be separated mechanically from the contents of the stomach, or from this organ itself. In a case of homicidal poisoning of a young actress in Berlin, the stomach was empty, there was no unusual smell, and only a few, suspicious, yellowish spots in the stomach, with no inflammation. In consequence of suspicions of the cause of death being aroused, by the fact of the husband of the deceased having shortly before purchased phosphorus paste (under a special permit), the stomach was submitted to a chemical examination. Cut into pieces, and warmed in a dark place over a spirit lamp, several shining points were observed in it, and afterwards, by directing a fine stream upon pieces of the stomach, removing fatty matters by boiling, and afterwards quickly cooling, the phosphorus was obtained in a globule of the size of the head of a pin.[50]

In another case of attempted poisoning, a woman prepared some soup for her husband. After he had taken a few spoonfuls, he was seized with pain in the stomach. In the evening his wife again pressed him to eat some more of it; but his suspicions were awakened, when, on taking it out of the warm and dark oven in which it had been put away, he observed that it was luminous.

The bowl was therefore sent to the magistrate. On uncovering it, white vapors, with a penetrating odor, proceeded from it. When the contents were poured out on an evaporating dish, a transparent, shining globule was observed at the bottom, and afterwards several more, which, when rubbed between the fingers, became luminous, and gave off white vapors. On boiling the soup over a spirit lamp, bubbles rose to the surface, which inflamed spontaneously.[51]

[50] Schacht, Casper's Vierteljahrs. 1852, April.

[51] Henke's Zeitschrift, E. H. Bd. XXVI. p. 173.

The detection of phosphorus is, however, seldom so easy. Being mostly taken, finely divided in the form of paste, and being not always rapidly fatal, it may be either removed by vomiting, or exist in too small quantity to be recognized with certainty. Nevertheless, as we have before stated, it has been detected in the body fourteen days after death. In another case, it was found ten days after death.

Dr. V. Elvers[52] reports a case in which free phosphorus was detected in the body of a woman eight weeks and two days after death. In this case 1½ grains of phosphorus were obtained from the intestines. This case is the longest on record of the detection of free phosphorus in the human body.

Various processes have been recommended for the isolation of phosphorus from organic mixtures. The best method is that employed by Mitscherlich, which consists in distilling the suspected material, previously diluted with water, if necessary, to the proper consistence, and acidulated with tartaric acid in a special apparatus called Mitscherlich's apparatus. In this apparatus the liquid, as above prepared, is contained in a flask, and, on being heated, its vapors rise through a small glass tube, at first vertically, then horizontally, and finally downwards. The descending limb of the tube is enveloped to a certain height with a cold water condenser, penetrating which, it passes into a closed vessel below. As the vapors, in coming over, reach the cool portion of the tube, they are condensed, and become luminous in the dark. By this method, 5 ounces of a substance containing only 1/40 of a grain of phosphorus, afforded 3 ounces of distillate in the course of half an hour, during all of which time the luminous zone was visible.

If the mixture, subjected to distillation, contain alcohol, ether, or, oil of turpentine,[53] no luminosity would be observed so long as these substances distilled over. Alcohol and ether, being very volatile, would soon be separated, and the light would then appear; this would not be the case with oil of turpentine, which substance would rarely be present in a medico-legal examination, unless it had been used as an antidote. Neither tartar emetic, magnesia, iron oxid, musk, castor, opium, albumen, any of the metallic salts, volatile, organic acids, nor free acids would interfere with the luminosity. Iodin, calomel, cor-

[52] Vierteljahrsschrift für gericht. Med. XXV. 1876, p. 25.

[53] During the last few years many cases have been reported in the different medical journals, where the oil of turpentine has been successfully used as an antidote for phosphorus poisoning. One of these cases is reported in detail by Andant in the Bulletin Générale de Thérapeutique, tome LXXVIII, p. 169.

rosive sublimate in large quantity, metallic sulphids, and particularly oil of wormseed, will interfere with the luminosity.[54]

If the luminosity of the phosphorus fails to appear when, approximately, one third of the contents of the flask has been distilled over, the apparatus is disconnected at the condenser end, and, in its place is connected a U tube filled with silver nitrate solution, and CO_2 passed through the whole apparatus for some time. If the silver nitrate remains unchanged, the absence of phosphorus may be inferred. If, however, the silver solution blackens, it must be tested for silver phosphid. An ordinary Marsh apparatus is used, and, after it is filled with hydrogen, the black, silver residue, which has been filtered and washed, is poured with some sulphuric acid into the generating bottle. The gas at the exit tube is lighted, and burns with a green flame, if the black substance was silver phosphid. The green flame is observed, with a spectroscope, to show the three lines in the green, in order to confirm the presence of phosphorus.

As a preliminary test, it is well to make use of Scherer's test, which depends upon the fact that phosphorus, phosphureted hydrogen, and phosphorous acid blacken nitrate of silver by reducing it to metallic silver. The best method of employing this reaction is that proposed by Hager,[55] which consists in treating a portion of the suspected substances, such as vomitus, contents of intestines, etc., with a little basic acetate of lead solution, in order to neutralize any sulphureted hydrogen which may be present, and placing the mixture in a tightly stoppered flask; a portion of it is placed in another flask, shaken vigorously with ether, and allowed to stand, tightly stoppered, for a few minutes; a piece of paper moistened with nitrate of silver solution should then be exposed to the action of the vapor in the upper portion of the flask, by fastening it into a slit made in the under surface of the stopper; the flask is then placed in a cool, dark place. If only a trace of free phosphorus be present, the silver paper will become blackened in from a few minutes to half an hour. If this preliminary test, which can be quickly performed, leads to a suspicion of the presence of phosphorus, it can be confirmed by distilling in Mitscherlich's apparatus as mentioned above. The various other processes for the isolation of phosphorus, some of which are of great value in special cases, need not be mentioned in detail here; the most important are those of Dusart,[56] and Lipowitz.[57]

[54] F. Hoffman, in London Chem. News, Jan. 1861.

[55] Pharmac. Centralhalle, 1879, No. 38.

[56] Jour. de Chimie Médicale, Sept. 1874.

[57] Poggendorff's Annal. der Physik u. Chem. XC. 1854, p. 600.

XIII. Mercury.

198. In general.— Metallic mercury is used in many of the arts. In medicine, in olden times, it was often used in the metallic form to remove intestinal obstructions, and if the accounts of such use are to be believed, there were rarely serious effects produced even after the ingestion of large quantities. The administration of mercury, however, at the present day, is held by the laity to be dangerous, though, apparently, for no reason.

The most common sources of poisoning by the compounds of mercury are the two chlorids, and, to a lesser extent, the oxids, sulphate, nitrate, and cyanid. Mercurous chlorid, $Hg_2\ Cl_2$, calomel, is a heavy, white, tasteless body, insoluble in water, but more or less acted upon by the digestive fluids, and if retained in the system, may produce severe symptoms.

Mecuric chlorid, $HgCl_2$, corrosive sublimate, is a white, crystalline substance, sublimes at 82.2°C, soluble in cold water, freely so in hot water, and still more easily in solutions of alkaline chlorids. It crystallizes in needles, octahedra or plates. It is soluble in alcohol, ether, and many other organic solvents. In the tissues, it forms an insoluble albuminate of mercury.

Mercuric oxid, HgO. Two modifications of this exist: one, a yellow, amorphous powder, obtained by precipitating mercuric chlorid with potassium hydrate; and the other, a red, crystalline body, prepared by heating mercuric nitrate. They are used extensively in ointments, the yellow one being more active than the red.

Mercuric cyanid, a white, colorless, crystalline body, soluble in water and alcohol. The aqueous solution has a bitter, metallic taste. The cases of poisoning by this salt of mercury have generally shown the symptoms of mercurial poisoning rather than those of the cyanid.

All forms of mercury are poisonous. Even metallic mercury itself, when in a finely divided form, as it is in mercurial ointment and in blue pill, can readily be absorbed from any surface, mucous membrane or skin, and give rise to symptoms of poisoning. Metallic mercury also frequently causes chronic poisoning, when inhaled in the form of vapor, since it is slowly volatile at the ordinary temperature of the atmosphere (above 40° F., 4.44°C.). There are large number of employments in which metallic mercury is used, and in which the workmen are exposed to the danger of inhaling its fumes, such as separating gold from its ores, and mirror, thermometer, and barometer manufacturing. All of the compounds of mercury, many of which

are used in medicine, also give rise to cases of poisoning. The most important of these are corrosive sublimate (mercuric chlorid, bichlorid of mercury) and calomel (mercurous chlorid, protochlorid of mercury); the latter is more frequently used in medicine, and has caused poisoning by being contaminated with corrosive sublimate, which is much more active since it is quite soluble. Calomel is also converted in the stomach by digestion into corrosive sublimate. Of the other sources of mercurial poisoning, we have the two iodids, mercuric and mercurous (red and yellow) iodids, both of which are quite largely used in medicine. The writer has recently met with a case of suicidal poisoning by the bisulphate of mercury. The two chlorids are, however, the most common sources of poisoning, since they can, as a rule, be easily obtained by the laity, corrosive sublimate being a common bedbug poison, and calomel being largely used in children's intestinal diseases. Corrosive sublimate is exceedingly active as a poison, on account of its ready solubility in water, alcohol, and ether, while calomel, being insoluble in these fluids, may often be given in very large amounts without producing serious symptoms. Most of the cases of poisoning are either accidental, owing to the ingestion of some preparation which was intended for external use, or owing to the use of impure (containing corrosive sublimate) calomel, or are suicidal. The exceedingly acrid, metallic, and styptic taste of the corrosive sublimate renders it unpopular with the criminal. Experiment has shown, also, that calomel may, when taken during digestion, be partially converted into corrosive sublimate, owing to the presence of free hydrochloric acid and the digestive ferment in the gastric juice at that time.

199. Preparations.— Calomel, or mild chlorid of mercury (hydrargyri chloridum mite, U. S. P.). Cyanid of mercury (hydrargyri cyanidum, U. S. P.). Red iodid, biniodid of mercury, or mercuric iodid (hydrargyri iodidum rubrum, U. S. P.). Green iodid of mercury, protoiodid of mercury, mercurous iodid (hydrargyri iodidum viride, U. S. P.). Yellow oxid of mercury, yellow mercuric oxid (hydrargyri oxidum flavum, U. S. P.). Red oxid of mercury, red precipitate, red mercuric oxid (hydrargyri oxidum rubrum, U. S. P.). Yellow subsulphate of mercury, basic mercuric sulphate, turpeth mineral (hydrargyri sulphas flava, U. S. P., 1870; hydrargyri subsulphas flavus, U. S. P., 1880). Mercuric sulphate (hydrargyri sulphas, Br.). Red sulphid of mercury, red mercuric sulphid, cinnabar, vermilion (hydrargyri sulphuretum rubrum, U. S. P., 1870; hydrargyri sulphidum rubrum, U. S. P., 1880). Mercury, quicksilver (hy-

drargyrum, U. S. P.). White precipitate, mercurammonium chlorid, ammoniated mercury (hydrargyrum ammoniatum, U. S. P.). Gray powder, mercury with chalk (hydrargyrum cum creta, U. S. P.). Sulpho-cyanid of mercury (hydrargyri sulpho-cyanidum). Nitrate of mercury (hydrargyri nitras). Mercuric methide. Emplastrum ammoniaci cum hydrargyro. Emplastrum hydrargyri. Massa hydrargyri, pilula hydrargyri (blue mass, blue pill). Unguentum hydrargyri. Unguentum hydrargyri ammoniati. Hydrargyri chloridum corrosivum (corrosive mercuric chlorid). Pilulæ antimonii compositæ. Pilulæ cathartieæ compositæ. Hydrargyri iodidum flavum. Liquor arseni et hydrargyri iodidi. Hydrargyri oxidum flavum (yellow mecuric oxid). Ointment of mecuric oxid. Unguentum hydrargyri oxidi rubri. Oleatum hydrargyri (oleate of mercury). Ointment of mercuric nitrate (citrine ointment). Liquor hydrargyri nitratis (solution of mecuric nitrate).

There are certain patent and quack medicines containing mercury and its preparations, among which might be mentioned, as common in England, Mordant's Norton's Drops, containing in solution a little corrosive sublimate colored with cochineal and flavored with ginger and gentian; Solomon's Anti-impetigines, also a solution of corrosive sublimate flavored and colored; Poor Man's Friend, nitrate of mercury ointment; Brown's lozenges, each lozenge contains ½ grain of calomel, 3½ grains of jalap resin extract with white sugar and tragacanth; Ching's worm lozenses, each lozenge contains 1 grain of calomel, sugar and tragacanth, colored with saffron.

There are also rat poisons, vermin destroyers, and fly paper which often contain mercury in the form of corrosive sublimate.

200. Statistics.— Death from accidental poisoning by mercury, as shown on Mr. Blyth's table, amounted in the ten years to 33, from suicide 24, from murder 3. In the twenty-six years recorded by the Massachusetts medical examiners, there were 10 deaths from accident or negligence and 26 from suicide by the use of corrosive sublimate (bichlorid of mercury). Witthaus and Becker have studied the records of 368 cases of poisoning from corrosive sublimate, collected from the literature of a hundred years just closed; of these, 22 occurred between 1870 and 1879, and 231 between 1879 and 1896. These authors justly refer to the increased use of corrosive sublimate solutions in antiseptic surgery; out of the last-named 231 cases of poisoning 141 occurred after irrigations of the vagina and urethra, of which 48 were fatal; of the 231 cases of poisoning, 172 were neither obstetric nor surgical, 70 were accidental, 47 suicidal, and 27

homicidal. In the above collection of cases by these writers, they note that, where fatal poisoning has occurred, the post-mortem appearances usually following mercurial poisoning were observed.

201. Symptoms of poisoning; absorption.—Mercury is easily absorbed—especially when associated with fat or oils—by the skin, and by all the mucous surfaces (whether introduced by the mouth, rectum, or vagina); mercury, in the state of vapor, is easily absorbed by the air passages; subcutaneous administration of mercury is rapidly absorbed. Mercury, in a finely divided form, may also be absorbed when rubbed into the skin; when combined with fat, its absorption by the skin may be so rapid as to cause fatal poisoning. Workmen in the quicksilver mines, and where gilding or silver plating on glass is employed in factories, suffer from mercurial poisoning, especially if the hands are not frequently washed. The symptoms of poisoning by vapor of mercury—and this is likely to be given off from metallic mercury at any temperature above 5° C.—are likely to occur, especially where mercury ores are in process of smelting.

We may consider corrosive sublimate as the type of mercurial compounds. Like many other poisons, corrosive sublimate or other mercurial preparations may cause death in a few hours, or the fatal issue may not occur for several days or weeks. Where death occurs within two or three days, the poisoning is called acute, and when it occurs later or within a few weeks, the poisoning is called subacute or chronic.

202. Acute poisoning.— The symptoms caused by acute poisoning are of the most urgent and alarming character, and generally supervene very soon after the corrosive sublimate is swallowed. At the moment of being swallowed, there is usually an involuntary constriction of the throat, which has, in many cases, prevented the whole of the draught from being taken, and a metallic taste in the mouth. Vomiting of a bloody and frothy liquid soon occurs, and continues throughout the case, attended with violent purging, but the latter symptom is sometimes absent. There is excessive pain in the abdomen, not always increased by pressure, and also burning and smarting in the throat. The general symptoms are, at first, those of febrile excitement, with great thirst, and are followed by exhaustion and collapse, a cold and clammy skin, small and frequent pulse, and difficult respiration. In most cases the urine is either entirely suppressed, or very scanty and difficult to void.

The duration of the case is very variable, sometimes terminating

in a few hours, but sometimes death may not occur until the third or fourth day. If life be prolonged sufficiently, the saliva should be carefully collected, and examined chemically for the presence of the poison. The urine should also be subjected to chemical analysis, because, if it is found therein, it shows that it has been absorbed by the general system, and is undergoing a natural process of elimination. The urine should also be examined, in the ordinary way, for the detection of disease of the kidneys; since, in all cases of acute poisoning by corrosive sublimate, when the poison is being eliminated by the kidneys, it irritates or inflames these organs, so that we are sure to find the urine albuminous and more or less bloody, according to the amount of irritation caused. In some cases the kidneys are acutely inflamed, and we find the condition known by physicians as acute nephritis or acute Bright's disease. When there is diarrhea, as is usually the case, the dejections are very painful, frequent, and bloody.

The symptoms of poisoning by corrosive sublimate more closely resemble those of arsenic than of any other poison, but may be distinguished, from the fact that the local action of the mercuric chlorid is corrosive, while that of arsenic is irritant; in the former, the animal tissues, brought in contact with the corrosive, show a whitish or grayish eschar with swelling, not unlike the effects produced by nitrate of silver. Occasionally, however, the corrosive action on the lips and inside of the mouth may be absent. Occasionally, also, the symptom of suppression of urine may be absent.[58] Another distinguishing feature, though, as will be seen farther, one not to be relied upon, is that corrosive sublimate, as well as all mercurials, usually cause salivation, or ptyalism, as an early symptom. Generally, salivation occurs within the first three or four days; but, in one case on record,[59] it occurred within the first four hours, and in another within one and a half hours.[60] Occasionally, this symptom is very much delayed, and in a case reported by Dr. Venables,[61] where two drams (7.77 gm.) caused death in eight days, no salivation occurred. The amount of salivation also varies greatly in different cases, where similar doses are taken; but it should be noted that salivation may occur without mercury, since, in one case reported by Dr. A. Farre,[62] in a pregnant woman who had taken no mercury, saliva was secreted to the amount of 3 pints a day, but in this case there was no swelling of the salivary

[58] Boston Med. and Surg. Jour. 1879, Pt. 2. pp. 781–783, reported by Drs. Schenk and Post.

[59] Guy's Hospital Reports, 1843.

[60] Med. Gazette, 1843.

[61] Taylor, op. cit. p. 356.

[62] Trans. Obstet. Soc. Vol. XV. 1874. p. 222.

glands nor fetid breath. In cases of salivation caused by mercury, these glands are usually swollen, and there is associated with the salivation an intense fetor, and ulcerated condition of the inside of the mouth, and, as a consequence of this condition, the teeth may loosen and fall out. According to Taylor,[63] salivation may occur after the use of gold, copper, bismuth, lead, antimony, iodin, iodid of potassium, croton oil, opium, prussic acid, carbolic acid,[64] sulphuric acid, arsenic, colchicum, foxglove or digitalis, and cantharides. Ulcerated gums and salivation may occasionally coexist in the salivation caused by arsenic and bismuth, but a chemical analysis of the saliva will reveal the nature of the poison. In salivation caused by mercury, the presence of this poison may be chemically detected in the salivary secretion, because it is eliminated from the system with this secretion, as well as by the intestines and by the kidneys. Salivation by mercurial preparations is not so common in the young as in the adult.[65] Dr. Craigie and Sir R. Christison state[66] that in the granular disease of the kidneys a small amount of mercury will induce salivation very readily. Several instances are detailed by Taylor, in which death was apparently caused by profuse salivation even after the use of medicinal doses of calomel. It may not be improbable that in these cases the death was due to the fact that the unhealthiness of the kidneys may have prevented a sufficient elimination of the poison; at any rate, it is important, in deaths caused by salivation from small doses of mercury, that the post-mortem appearances of the kidneys should be presented in evidence.

203. Chronic poisoning.— The above account of symptoms is mostly concerned with cases of acute poisoning by corrosive sublimate, with the exception of the account of salivation, which is more common with subacute and chronic poisoning than with acute. In the case of slow or subacute or chronic poisoning by mercurial preparations, the symptoms of local, corrosive action on the primæ viæ may be absent, and the effects of slow poisoning only may be observed. In cases of slow poisoning, the symptoms of continuous use of small, noncorrosive doses will be those of salivation, as above described, with the appearance of a blue line on the margin of the gums not unlike that caused by lead poisoning. Usually the first symptom of chronic poisoning, caused by the repeated or continuous absorption of small amounts of mercury, is a tenderness of the teeth, which is especially noticeable

[63] Op. cit.
[64] Guy's Hospital Reports, 1870, p. 533.
[65] Dub. Med. Press, May 12, 1847.
[66] Taylor, op. cit.

on bringing the jaws forcibly together, so that physicians, when prescribing a mercurial in cases of syphilis, should always direct their patients to do this; and if they notice any such tenderness to omit the medicine for a short time; sometimes a little looseness of the bowels will occur before the tenderness of the teeth. Of the other premonitory symptoms of chronic poisoning, we have great pallor and excessive weakness; this weakness progresses steadily, affecting first the muscles of the upper limbs, after a longer period extending to the lower limbs, and finally, if the patient lives long enough, it ends in general palsy or trembling of all of the muscles. Colicky pains in the abdominal region, associated with nausea, vomiting, general restlessness, and a feeling of malaise, may also be present. The salivary glands become tender and swollen, and the mouth inflamed and ulcerated. The patient may also be troubled with difficult breathing and deglutition. The serious salivation, or ptyalism, is the most prominent symptom, and may threaten or cause death. Dr. Johnson[67] reports a case where 3 grains (0.194 gm.) of gray powder (mercury with chalk, hydrargyrum cum creta), which contains 50 to 60 per cent of mercury, caused salivation, and Sir R. Christison[68] reports another case, in which 2 grains (0.130 gm.) of calomel (mild chlorid of mercury) destroyed life by salivation. Taylor reports a case[69] in which profuse salivation and sloughing ulcerations caused fatal poisoning in a child of eight years, death following four days after taking three doses in one week, which amounted to a total only of 16 grains (1.04 gm.)[70] In still another case, six doses in three days, amounting to a total of 18 grains (1.17 gm.) caused, in a girl of nineteen years, severe salivation, loosening of the teeth, and death in twelve days. It is also sufficiently well shown that salivation may appear, disappear for several months (three to six), and then reappear in an aggravated form, without any mercurial compound having been taken in the mean time. In all these cases it is very important, as has before been observed, to examine the condition of the kidneys. There is one curious fact to be observed in regard to mercurial treatment, which may be of medico-legal importance,—that the system can easily and more rapidly eliminate one large dose of mercury than the continuous administration of small doses frequently repeated; and that habitual constipation, associated with continuous, mercurial treatment, seriously obstructs the elimination of the drug. This fact is not unreasonable, since the mucous glands in the intes-

[67] Sanitary Record, July, 1874.
[68] Taylor, op. cit.
[69] Op. cit. p. 357.
[70] Lancet, Dec. 20, 1851, p. 579.

tinal canal lend material assistance in the elimination of mercury, and large doses of mercury generally induce diarrhea.

It should be observed here that mercurial inunctions (where mercury is rubbed up with fat, and thus becomes subdivided into very many minute globules) cause a very ready absorption of mercury through the skin, and the broken skin of the hands in contact with the poison will easily allow its absorption. It is also well known that the presence of a large body of a mercurial preparation, in badly ventilated apartments, will readily allow absorption of the poison, from the distribution of mercurial vapor in the air, and its inhalation by the lungs. Solutions of corrosive sublimate were formerly used by surgeons to purify their hands; Taylor[71] gives instances where mercurial poisoning was thus induced. This writer also enumerates several instances where ointments or lotions, sold by quacks as secret remedies for diseases of the skin, have induced fatal poisoning. One case of especial value, that reported by Mr. Annan, illustrates this danger. A shepherd used a lotion of 2 drams of corrosive sublimate, dissolved in 20 ounces of water, to wash sheep affected with some skin disease, and died from the secondary effects of mercurial poisoning; and of ten sheep thus treated two died shortly after the application of the lotion.

As before observed, death may occur in a few days in some cases, and in others be prolonged beyond a week. In a case reported by Dr. Coale, death took place on the eleventh day; and in another, by Dr. Jackson, on the thirteenth day.[72] Death may not occur until later from the consecutive effects. Such was the case in an instance reported by Dr. Ware, where the patient died with profuse discharge of mucus and bloody matters from the bowels on the fifteenth day.[73]

A few other cases of death from the external application of corrosive sublimate are on record. In one, the subject was a child, and the severest constitutional effects were produced. It died in about a week.[74] In two others, also children, of seven and eleven years respectively, an ointment, composed of 2 drams of corrosive sublimate to 1 ounce of tallow, was rubbed into the scalp. The children were affected with porrigo favosa. Excessive suffering was the almost immediate consequence, and in forty minutes they were completely delirious. They vomited continually a green-colored matter, had great pain in the bowels, with diarrhea and bloody stools. In the youngest there was complete suppression of urine. Death occurred in one on

[71] Op. cit.
[72] Am. Jour. Med. Sci. Jan. 1851.
[73] *Ibid.*
[74] *Ibid.* July, 1844, p. 259.

the seventh, in the other on the ninth day. There was no ptyalism.[75] In a case in which from 15 to 20 grains of corrosive sublimate were injected into the vagina, there occurred, besides local symptoms, vomiting, bloody purging, coldness of the extremities, spasms of the fingers and toes, and salivation. Recovery took place.[76]

"The symptoms of mercurial poisoning are of incontestable value as evidence, and more so, perhaps, than for any other poison of the inorganic group; the clinical record has here a certain signification, and allows of an almost certain diagnosis.[77] There is no disease which, in the acute form, has such a fatal result, consequent upon the specific buccal lesions; and for the chronic form, the cachexia, which constitutes the common ground of so many diseases, imprints in this case a particular character to such nervous phenomena as the tremblings, partial paralysis, and pains in the bones. Syphilis alone can be confounded with chronic mercurial poisoning." [78]

The subject of chronic poisoning by mercurial preparations, and the discussion of questions arising out of the specific effects of them upon the system, involve too many considerations to be profitably introduced here. We therefore refer the reader, for information on these points, to Dr. Christison's treatise, and to the standard works on pathology and the practice of medicine.

We cannot forbear, however, to call the reader's attention more particularly to that form of disease known under the name of cancrum oris, gangrænopsis, and mercurial sore mouth, especially in children.

That death may occasionally result from the action of mercury upon the mouth, there can be no doubt. It is extremely important, however, to know if this can be distinguished from those forms of inflammation and gangrene of some portion of the buccal cavity which are the result of certain depressed and diseased conditions of the system, independent of the action of mercury; and also, whether mercury, given to a patient whose vital force is thus reduced, and whose blood is already depraved, may not be the exciting cause by which the tendency to gangrenous ulceration becomes developed. It is much to be feared that the inappropriate administration of this drug, has, in some cases, been followed by serious, and even fatal, disorganization of the mouth; while, on the other hand, physicians may be unjustly blamed for consequences which were really not the result of

[75] De Ricci, Dub. Quart. Jour. Aug. 1854.

[76] Butcher, Dub. Quart. Jour. Feb. 1856, p. 242.

[77] Tardieu, op. cit. p. 571.

[78] *Ibid.*

their imprudence, but of other causes which they were unable to control.

We propose first, by a few cases, to contrast the appearances presented by the effects of mercury on the mouth, with those which are due to disease.

A boy about ten years old, supposed to be suffering with bilious colic, was given 20 grains of calomel, which purged him in four hours; he took, in twenty-four hours, 10 grains more with the same effect, but without much relief. On the morning of the fourth day (medical treatment having been continued, but no calomel or any of the acids used) all the symptoms of the early stage of ptyalism set in; the inflammation and swelling of the salivary glands rapidly increased, so that, by the day following, there was a general swelling of all the soft parts of the face usually affected by severe ptyalism. On the next morning, a small, gangrenous spot, of a dark brown color, was discovered on the middle and inner surface of the lower lip, which rapidly spread until the seventh day. At this time, the entire lower lip, the inside of both cheeks, and the surface of the tongue, were completely gangrenous, the lower lip and the tip of the tongue were wanting, having been destroyed by ulceration. The ptyalism increased, a stream of viscid saliva was constantly running out of the mouth, and the patient presented a most piteous spectacle. The breath was very fetid and offensive. Death occurred on the twelfth day. In another case, a little girl ten years old, who received a fracture of the head from a fall, was given some calomel for the purpose of opening the bowels, but it did not operate. Her mouth became sore, and got rapidly worse, notwithstanding every effort was made to relieve it. The throat and face became immensely swollen; the teeth became loose and several came out, and the whole inside of the mouth, tongue and all, had a very black appearance, emitting a constant flow of a dark, putrid saliva of intolerable fetor. The greater part of the mouth and tongue mortified, and part of the tongue, the under lip, and part of one side of the face sloughed off, presenting a most horrible spectacle, and one exquisitely distressing to the parents and friends of the little patient—the more so as the child continued to live some days after these parts had become detached.[79]

That form of disease due to the action of mercury upon a depraved constitution may be illustrated by the following cases:[80] A boy, aged thirteen years, after suffering from influenza and partially recovering,

[79] Boston Med. and Surg. Jour. Vol. XXXII. pp. 459 and 517.

[80] *Ibid.* p. 342.

was attacked with gastroenteritis, from over-indulgence in animal food. The bowels were moved daily with about 2 grains of calomel, followed by a teaspoonful of castor oil, when necessary. He improved under this treatment; but, about three weeks after the commencement of his sickness, it was discovered that mortification had commenced under the tongue, near the third molar tooth, on the left side; it extended around all the molar teeth of that side, embracing the gum and a portion of the cheek. The cheek was slightly swollen, and the left eye was opened with some difficulty. The mortification spread rapidly, notwithstanding the use of caustics, a dark spot appeared on the outside of the cheek, and the patient died on the third day. It is stated that the boy had dug out a piece of a tooth with a knife, a few days before the mortification began, at the place where it commenced. About 20 grains of calomel were taken during the first week of the disease, and none afterwards. The glands were not affected; and the mouth, before the mortification commenced, presented a healthy appearance.

Dr. S. Jackson (late of Northumberland) says: "I applied mercurial ointment to the face of a child, about three years old, to prevent the pitting of confluent smallpox; in a few days the gums were swollen and the teeth loosened, but only on the side upon which the patient continually lay. The gums soon mortified, the gangrene spread to the cheek, bringing on a fair case of gangrænopsis, and she died of her twofold disease in a very few days. This," he says, "was not a case of salivation, for the other side of the jaw remained sound, and the teeth on that side firm in their sockets."[81] In the same manner were fatal ulceration and gangrene developed in a case reported by Dr. Marshall Hall. A child four years of age, with whooping cough, took, according to a prescription furnished from a dispensary, 3 grains of calomel, on the 29th of October, and the same dose four times thereafter until the 7th of November. About this time the right cheek became much swollen, and there was great difficulty in opening the mouth, with very offensive breath. The gums and inside of the cheek became ulcerated, and on the 16th a sphacelus appeared on the right cheek, of about the size of a shilling, which rapidly extended to the size of a crown. The child continued to get worse, and died on the 23d. On post-mortem examination, there was found pleuropneumonia of the lower lobe of the right lung; there was an extensive eschar in the right cheek; its size, externally, was 2½ inches

[81] Trans. Coll. of Phys. Phila. U. S.
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in length by 1½ in breadth. It penetrated through the entire cheek, and occupied an equally extensive space on its internal surface; the contiguous gum was in a similar state of sloughing, the alveolar processes were denuded, one or two teeth had disappeared, and several adjacent ones were loose. On the left side there was incipient gangrene of the cheek internally, and also of the contiguous gum, and the teeth were loose. The rest of the mouth was not affected.

Cases of true gangrene of the face, however, have a different origin and course. A single example will suffice. It is taken, also, from Dr. M. Hall's Observations in Medicine. A little girl, aged three and a half years, had been affected with fever about fourteen days, and was apparently convalescent, when the left side of the face and lips was observed to be swollen, and to have a red and glistening appearance. About the same period, three spots were observed, one on the gum of the lower jaw, and the other two on the left cheek. These spots became dark-colored, and gradually spread. A slough separated from the cheek, and exposed the inside of the mouth. The contiguous teeth fell out. The breath and the exhalation from the ulcer were extremely offensive. The child lingered about fourteen days, and sank gradually.

Having thus seen the various forms of disease of the mouth which may give rise to a suspicion of poisoning by mercurial preparations, it only remains for us to point out the means by which the symptoms, really caused by such preparations, may be distinguished from others which are spontaneous in their origin.

All authors agree that mercury does not produce salivation in children, as readily as in adults. Dr. Clarke says that, although he has prescribed mercury in very large quantities in a great number of cases, he never produced salivation, except in three instances, in any child under three years of age. Dr. Warren, of Boston, observes that he has never known an infant to be salivated, notwithstanding he has given in some cases, large quantities, with this view. Mr. Colles, of Dublin, says: "No man in the present day requires to be told that mercury never does produce ptyalism or swelling and ulceration of the gums in infants." Drs. Evanson and Maunsell say: "Mercury does not seem capable of salivating an infant; we have never seen it do so, nor are we aware of any such case being on record."[82] Dr. West, of London, says: "In infants under five years of age, the gums hardly ever become affected by mercury, though most energetic-

[82] Beck's Essays on Infant Therapeutics, p. 48.

ally employed; and it has never yet occurred to me to meet with an instance of profuse salivation or dangerous ulceration of the gums. Such accidents, however, do now and then occur, and have been known to terminate in fatal gangrene of the cheek, or necrosis of the jaw." Yet, when salivation does occur, there is quite sufficient testimony, which it is unnecessary to quote, that the most disastrous consequences may follow. In this fact, however, appears to lie the great distinction between the disease resulting exclusively from the use of mercury, and that which is spontaneous or merely called into action by it. Dr. Hall says: "It is well known to every observer that the effect of calomel, when it does take place, is uniformly diffused over the gums, tongue, and internal parts of the cheek." Further, "it is diffused, and is totally different in many respects from the circumscribed form of the gangræna oris." In other words, the mercurial disease commences in the gums and tongue; they swell, ulcerate, and slough, and the disease may then extend itself to the lips and cheek. The disease is, therefore, different in its early manifestations, is attended with salivation, is slower in its progress, and at first confined to parts which in true gangrænopsis are only secondarily affected.

We copy the following description of gangrænopsis from the monograph of Dr. Jackson, to which we have already referred:

"I. The gangrænopsis attacks the cheek, the lip, or the nose, sometimes the fauces; most frequently in children, but sometimes in adults.

"II. It begins in those soft parts, and never in the maxilla, often where no mercury has been used, in a debilitated and febrile state of the system, as in idiopathic fevers and dysentery. Van Swieten saw it in scurvy, and Huxham in measles. Dr. Marshall Hall says (p. 178): 'In all the cases which came to my knowledge, this affection has been preceded by fever, acute disorder of the digestive organs, inflammation of the lungs, variola, rubeola, or scarlatina.' An exhausted state of the vitality, with cachectic fever, is, therefore, the predisposing cause.

"III. The exciting cause is any injury done to the parts. I saw it evidently started in two cases by the child's lying continually on one side, with a hand under the cheek, thus pressing the mucous membrane against the molar teeth; a protuberance of this membrane, being caught between the teeth, was continually bruised, and a point of gangrene was thus established in an exanimate state of the whole system.

"IV. It is sometimes the result of severe cases of cancrum oris, the irritation spreading from the gums to the cheek."

It is well known that cancrum oris, and the gangrene which attacks the cheek, often occur in cases where no mercury has been given. We think that there is between these two little pathological difference; the most tangible distinction being, as it appears to us, that the canker sore mouth of children (as it is called) sometimes prevails endemically in low, unhealthy situations, and among the poorer classes, being frequently seen in the hospitals for children, and occurring without being necessarily preceded by disease; whereas, the gangrene of the cheek is commonly a sequel of exanthematous or other prostrating diseases, such as measles, scarlet fever, and diphtheria. Both are allied closely to the gangrene of the genitals in female children, elsewhere referred to. (See RAPE, Vol. III., § 187e.)

We have carefully given the various opinions of physicians upon the subject of mercurial sore mouth in children, and the well-known fact that sore mouth, unprovoked by mercury, may exist among infants. Though the question appears from the above historical *résumé* to be somewhat complicated, it need produce no serious embarrassment in any given case, since all doubts of the cause in a suspected case of poisoning may be set at rest by a chemical analysis of the child's saliva or urine, when the presence of mercury will be declared, if that is the cause. On the other hand, the fact cannot be denied that many medical practitioners use mercury in cases even of sore mouth, yet this matter can be settled by evidence adduced to show, by the physician's testimony, that the sore mouth existed prior to the institution of mercurial treatment. It is a mooted question among physicians whether mercurial treatment in serious ulcerations and gangrenous affections of the mouth and throat is a wise measure; for, at a recent discussion in a medical society, in the presence of the writer, he was astonished to learn that practitioners of good reputation advised the use of mercury in putrid sore throat and diphtheritic inflammations. The symptoms and course of gangrene in the mouth are described by Niemeyer,[83] on the authority of Barthez and Rilliet.[84] The gangrene commences, usually without pain, on the inner surface of the oral mucous membrane; a soft, regular, circumscribed edema occurs in the affected cheek and lips, and gradually spreads. A hard, round nucleus forms in the center, over which the skin appears shining, pale, or mottled violet. Even when

[83] Text-book of Practical Medicine, New York translation, 1869.

[84] Maladies des enfants, Paris, pp. 359 *et seq.*

the inside of the cheeks and a great portion of the gums have become gangrenous, the child often sits quietly in bed. A sanguineous, or even black, saliva runs out of its mouth; but it plays, demands food, which it takes eagerly, and with the food swallows the sloughs that fall off from the gangrenous patches. At the same time the skin is pale and cool, the pulse small and moderately frequent, and there is delirium at night. Occasionally, mostly at the fifth or sixth day of the disease, a circumscribed, dry, black slough forms on the cheeks or lower lip; this may increase daily, till it affects half the face. Occasionally, even at this stage, the child is tolerably strong, will ask for food while pulling gangrenous patches from its mouth. The child's appearance is repulsive, as the slough separates from the underlying tissue, and the jawbone and loosened teeth show frightfully. The odor in the exhaled breath is offensive, the patient grows weaker and weaker and diarrhea supervenes. The patient may die from exhaustion without any febrile disturbance, or may even recover from the extremity of the disease; a healthy suppuration taking the place of the gangrenous sloughs. We have taken great pains to give a detailed description of this gangrænopsis or canker or sore mouth, since unjustly many children have been supposed to have been poisoned by mercury, when this spontaneous disease is present.

The description of mercurial stomatitis is presented in an article by Chauffard,[85] from which we take the following abstract: "The commencement of mercurial stomatitis or sore mouth is marked by a dryness of the mouth, attended with heat and pain, and is accompanied by a true inflammation of the mucous glands. The breath has a peculiarly offensive odor difficult to describe, but easily recognized by one who has once perceived it, and, simultaneously with this fetor, the subject complains of a disagreeable taste, nauseating and metallic. The teeth feel as if they were 'on edge,' and become painful, and seem lengthened on account of the swelling and inflammation of the underlying periosteum of the alveolar process. Very soon the movements of the mouth in efforts of mastication become painful, but this is soon replaced by a pain which is restricted to the teeth and cheeks, though sometimes it is likewise felt at the angle of the jaw, and extends to the side of the face, simulating facial neuralgia. The pain soon becomes so intense that only liquids are swallowed, the patient having some difficulty in separating his jaws on account of a sort of reflective tetanus. The initial dryness of the mouth is of short duration, and

[85] Nouveau Dict. de Méd. et Chirurgie, tome XXXIII. Paris, 1882.

soon is replaced by the opposite phenomenon of salivation. In light cases this salivation is distressing and painful, but may not be excessive; the patient may spit during the day, and drivel (drool) during the night, and this may be the whole extent of the salivation. In severe cases, however, the ptyalism may assume remarkable proportions, there being a continuous dribble, or excessive flow of a clear or grayish saliva, having an insipid and fetid odor (Grisolle). Several quarts may flow during twenty-four hours, and the most simple chemical test will show the presence of mercury. This excessive secretion exhausts the patient, troubles his sleep, and may even, as before remarked, cause his death. During the presence of the above symptoms, a physical examination will reveal an intense inflammation in the mouth. In light cases, the gums are swollen and tumefied, and of a livid, violet shade; where the gums naturally should join the teeth, they are separated, and show a lively red or whitish line, ulcerated and bleeding at the slightest touch. The teeth thus exposed seem lengthened, loose, painful on pressure, and are covered with a grayish envelope. Simultaneously with these physical appearances, the inflammation will extend over the whole cavity of the mouth—on the margin of the tongue, the internal aspect of the cheeks, the posterior surface of the lips. The mucous membrane assumes a vivid red color, is infiltrated, covered with grayish or opalescent, epithelial scales, and shows the imprint of the teeth and the raised impress of their furrows. Even in light cases the tongue participates in the inflammatory action of the mouth, the evidence of its swollen, inflamed condition corresponding with the stomatitis, which is a very certain evidence of its mercurial cause."

In severe cases of mercurial stomatitis or sore mouth, a copious and even bloody salivation is accompanied by very active pain in the submaxillary and other salivary glands. The teeth loosen and fall out in a few days' time, sometimes making the patient actually toothless. Along with these phenomena, the grayish patches of the inflammation of the mouth invade the inner aspect of the cheeks, the margins and upper surface of the tongue, and below it, may be found fissures and more or less deep ulcerations of the mucous membrane. The swelling of the tongue becomes so extensive that it may fill up the whole cavity of the mouth, and may even cause it to project. Respiration becomes difficult, on account of the swollen and infiltrated tissues, and may cause a true mechanical asphyxia. The inflammation of the tongue may extend backward into the pharynx and throat, and may cause deafness, as well as pain in the ears, from

extension of the trouble through the Eustachian tubes; its extension into the throat may cause an attack of suffocation from edema of the glottis.

In the most severe cases, but rather exceptionally, gangrenous patches of the surface of the mouth may be observed, necrosis of the jawbones, a general restlessness accompanied by fever and headache. The patient can neither eat nor sleep, and may pass into a state of cachexia which may become fatal. The duration of mercurial sore mouth is entirely dependent upon its intensity, and light cases may recover in eight or ten days. In cases of medium intensity, on the contrary, the salivation may resist the most rational treatment, and persist obstinately during weeks and even months. Convalescence is always slow, and it is seldom that patients can chew solid food for a long time without provoking pain and swelling of the gums. Finally, the extreme cases, when cured, are left in a pitiable condition, feeble, cachectic, and often disfigured by frightful scars caused by the loss of the teeth, or decay (necrosis) of the jawbones.

The differential diagnosis between true gangrene, stomatitis or sore mouth, and mercurial stomatitis is easy of recognition, even without the aid of a chemical test for the presence of mercury in the saliva. A comparison of the symptoms above given, in either case, shows a marked difference between the affections. In natural gangrene, the disease begins by ulceration, without inflammatory action of the mucous glands and without pain, while, in mercurial stomatitis both are present. The other points of difference are presented in such detail that it would be wearisome to repeat them. It is evident, we think, from what has been said, that the diagnosis of the cause of these various forms of disease is not difficult. It depends chiefly upon the possibility of ascertaining the manner in which the disease first manifested itself,—whether by swelling and ulceration of the gums generally, with an increased flow of saliva; or, whether it commenced in the mouth or cheek with a hard red swelling, rapidly running into gangrene. The character of the disease under which the child was suffering, and its hygienic conditions, must also be known.

If an opinion is required only after death, or at an advanced period of the disease, it may be impossible to know whether it can be ascribed to mercury, or whether, in case it is known that mercury has been exhibited, it can be fairly attributed to it. Dr. Taylor does not admit the validity of the criterion that mercurial poisoning can be known by the uniform diffusion of the disease over the gums, tongue, and internal parts of the cheek, as advocated by Dr. Hall. And Dr.

Christison, he says, has recorded a case in which, although the gangrene resulted from mercury, it was observed to occur on the skin near the mouth, on each side, whence it spread over the whole cheek, and destroyed life in eight days.[86] He also gives a case in which a charge was made against a medical practitioner, of having caused the death of a child aged four years by administering an overdose of some mercurial preparation. The child was laboring under whooping cough, and some medicine was prescribed; on the fourth day, the child complained of soreness of the mouth, the teeth became loose and fell out, the tongue and cheek were very much swollen, and the child died in the course of a few days from gangrene in the left cheek. The answer to the charge was that not a particle of mercury had been exhibited; a fact clearly proved by the production of the prescription book of the medical attendant.

Chlorate of potassium is an almost certain remedy against cancrum oris, and also is the best treatment for the mercurial sore mouth.

The inhalation of mercurial vapor, derived from breathing an atmosphere, in warm weather, in confined apartments, very rapidly produces symptoms of mercurial poisoning. Besides the symptoms enumerated above, there have been observed affections of the eyes such as conjunctivitis, caries of the teeth and of the jawbones, restlessness, sleeplessness, headache, noises in the ears, giddiness, fainting, etc. In these cases the muscular tremors are suspended during sleep. Phthisis has long been known to be a sequela of chronic mercurial poisoning. It should also be noted that persons, suffering from chronic disease of the kidneys, cannot bear as large doses of mercury as when these kidneys are in a healthy, normal condition.

It must be admitted that, in these cases of renal disease, whether acute or chronic, the effects of mercurialization upon the system will be greatly increased, and may even cause serious, if not fatal, consequences. The evidence, therefore, of albumin and casts of the renal tubes being present in the urine during life, as well as the post-mortem changes of the kidney tissue, will throw light upon the consequences of the administration, even of small doses of mercury; and this evidence should not be omitted in a medico-legal investigation.

204. Lethal dose.— It should be noted that the lethal dose of mercurial salts varies, sometimes from idiosyncrasy, sometimes from certain diseases of the organs of elimination, as mentioned above. It should also be noted that where a cathartic action follows a dose of

[86] Op. cit. p. 319.

some of the forms of mercury, it may pass out of the system so rapidly as, sometimes, to escape absorption. If, however, corrosive sublimate should produce its corrosive action upon the primæ viæ, the general lesions, localized in the stomach and intestines, may be so serious as to endanger life. Fatal results may therefore follow from the acute form of poisoning, and even in the subacute and its chronic forms, where these lesions may interfere with digestion. It is reasonable to conclude, on the authority of Taylor and others, that fatal poisoning could follow the ingestion of 3 to 5 grains (about 0.2 to 0.325 gm.) of corrosive sublimate. If prompt treatment, after a poisonous dose, is followed, it may prevent fatal poisoning even from much larger doses, 1.5 drams (6 gm.). Recently, Mann[87] has reported several deaths from white precipitate, mercurammonium chlorid; one of these cases, from 35 grains (about 2.3 gm.). Red precipate, red oxid of mercury (U. S. P.), in doses of two or more drams (7.7 gm.) has caused serious inflammatory action upon the gastrointestinal tract.[88] The ingestion of acid nitrate of mercury, (liquor hydrargyri nitratis U. S. P.), which should be used only as an external irritant, has resulted in fatal poisoning when swallowed.[89] Turpeth mineral, (hydrargyri sulphas flavus U. S. P. 1890), has been followed in one case after two doses of 3 grains (0.2 gm.) by death.

The smallest quantity capable of destroying life is not ascertained with precision. Children have been killed by 3 grains. Taylor considers that the average fatal dose may not differ widely from that of arsenic, *i. e.*, 2 or 3 grains (0.13 or 2 gm.), but, like doses of arsenic, a fatal dose, in one case, has proved innocuous in others, and in cases of disease like syphilis, enormous doses have been given with impunity for one and two years continuously; in this, the syphilitic poison seems to offset the dangers from the metallic poison. Corrosive sublimate is not, however, the most common form of prescribing mercury in syphilis, practitioners preferring the biniodid, or protochlorid of mercury, or by inunction.

In Dr. Coale's case, less than 10 grains (0.65 gm.) were swallowed. Dr. Frisselle reports a case which is remarkable for the indifference to the symptoms upon the part of the patient. A woman took a dram (3.9 gm.) of corrosive sublimate in solution. She was immediately seized with a burning sensation in the throat, and copious vomiting of a dark, frothy substance, which was followed in about an hour by purging, which continued till the next day. She still, however, at-

[87] Forensic Medicine and Toxicology, Phila. 1893.

[88] Ord. London Lancet, 1899.

[89] Med. and Surg. Rep. 1884.

tended to her domestic duties, and no remedies were given internally until thirty-six hours afterwards. She died on the sixth day, with vomiting of a dark grumous matter.[90]

"Persons have been known to recover, who have taken very large doses, when remedies have been timely administered, or when there was early vomiting. . . . A case of recovery, after 40 grains had been taken in whisky, under circumstances favorable to its fatal operation, *i. e.*, on an empty stomach, is recorded by Dr. Andrews, in Cormack's Journal, Feb. 1845, 102."[91] Poisoning by this agent is oftentimes extremely slow, and sometimes is not manifested for several months after exposure to mercurial vapors. Long before this period, the face becomes swollen, the complexion livid, all the functions languish, ending in hemorrhages from the mucous surfaces, and, from time to time, the patients are afflicted with diarrhea.

205. Treatment.— If vomiting occurs as an early symptom, white of egg and milk should be given immediately, in order to produce an incoluble compound with the albumen. Magnesia also forms a compound with corrosive sublimate, which is less injurious than the former.

The antidote to mercurial poisoning is albumen in a large amount of water (too much of the antidote causes a resolution of mercury). The albumen is precipitated in a solid form by the mercury, and this can then be ejected from the stomach; but if retained in an excess of albumen, its resolution may cause subsequent and subacute poisoning.[92]

206. Action.— The theory of the action of this poison has been explained by Dr. G. Sée.[93] As soon as the mercury is received into the blood, it is combined directly with the albumin of the plasma and the protein of the corpuscles; this is the cause of mercurial anemia. The white corpuscles, however, maintain their normal condition.

Though death may follow fatal poisoning in half an hour from serious injury to the primæ viæ, life may be prolonged for a few days up to ten days.

Immediately after swallowing a poisonous dose of bichlorid of mercury, there is experienced a strong, metallic taste, and a painful, spasmodic contraction in the throat. The mucous surfaces of the mouth and of the primæ viæ, coming in contact with this corrosive,

[90] Boston Med. and Surg. Jour. 1850, p. 279.

[91] Taylor, Am. ed. of 1875, p. 367.

[92] See the case referred to in Boston Med. and Surg. Jour. by Drs. Schenk and Post.

[93] Leçon de Pathologie Expérimentale, prémier fascicule, Paris. 1866, p. 275.

are shriveled and whitened, giving the appearance of the local application of lunar caustic (silver nitrate). The vapor of mercury, inhaled by animal life for a continuous time, produces a redness of the gums, which afterwards become swollen, and this effect is accompanied with loss of appetite; the coating of the eyelids and the eyes is also inflamed. The careless application of mercurial ointments and washes to sheep, used to destroy parasites, has resulted in the absorption of mercury either by the broken or unbroken skin; in these instances, sheep become emaciated, refuse to eat, and breathe distressingly with short, quick gasps.

Experiments on rabbits (which do not vomit), dogs, and warm-blooded animals, produce salivation and stomatitis, paralysis and the other nervous affections which we have indicated in the descriptions of poisoning. When absorbed into the system in man and lower animals, the action of corrosive sublimate apparently interferes with the functions of the cells lining the tubules of the kidneys, producing in them a condition called by Kobert, necrobiosis, destruction of life of their epithelium. The action of the corrosive agent seems to be exerted upon the whole intestinal tract,—especially in the lower parts of the smaller intestines, and also in the large intestines. Consequently, it may be inferred that the action of large or poisonous doses of the corrosive sublimate, by destroying the local functions of the cells in the eliminating organs of the large intestines and kidneys, would increase the action of this poison upon the tissues of the body. This action would account for the delay of the mortal symptoms, unless due to the immediate, early, fatal shock, from the destruction of the primæ viæ, exerted upon the nerve centers, thus inhibiting their functions. It will have been noticed that, unlike arsenic and some of the other poisons previously described, the fatal period is prolonged from twenty-four hours up to ten days.

207. Diagnosis.— It will be observed from the foregoing descriptions of mercurial poisoning that we have to make a differential diagnosis between arsenical poisoning, phosphorus poisoning, and nitrate of silver poisoning. Judging from the symptoms alone, apart from the local corrosive actions of these poisons, arsenic and phosphorus, as well as antimony, show profuse diarrhea, arsenic and antimony having the peculiar rice-water discharge which has been previously mentioned. Phosphorus usually gives off phosphorescent light and odor; arsenic, without phosphorescent light, gives out odor; both these poisons are followed by rapid, fatal symptoms; both these poisons have the peculiar, pustular eruptions, previously described

as upon the mucous membrane of the mouth and throat. The one important, diagnostic symptom attributed to mercury is the stomatitis salivation and the black-edges appearance on the gums; the presence of mercury in the fecal discharges, vomit, and urine, can be determined by analysis.

208. Post-mortem appearances.— In the case mentioned by Dr. Frisselle, no appreciable lesion was discovered. In one reported by Dr. Williams,[94] the stomach, which was contracted in the shape of a dumb-bell, presented patches of dotted injection of a bright crimson tint. There was no ulceration nor ecchymosis, but the mucous membrane was a little softened in the neighborhood of the most vivid red patches. Similar patches were seen throughout the small intestine. The bladder was contracted and contained about a dram of turbid urine. The other organs were healthy. The dose, in this case, was 30 grains (2 gm.) and the patient lived two days. In an instance reported by Dr. Herepath, the stomach seemed to have escaped the action of the poison, but the cæcum was of a deep, black-red color, and portions of it were in a sphacelated condition.[95] The mucous membrane of the mouth and fauces usually exhibits traces of the action of this corrosive poison, being changed to an ashy-blue color; but, in the first two cases here noted, and the following one, in which a dram (4 gm.) was taken, the mucous membrane of the mouth and œsophagus was perfectly healthy. The principal effects of the poison were observed in the stomach; its mucous and muscular tissues (beginning at the cardiac portion, 3½ inches in extent by about the same in breadth) were converted into a gangrenous mass, having a corroded, ragged appearance, of a dusky-brown color, approaching to black. The mucous coat, to some extent, around this gangrenous portion, was of a brownish-red color, but the lining membrane of the pyloric half of the stomach, except a few slightly red patches, was quite healthy. The bladder contained only half an ounce of urine, although none had been passed for twenty-four hours before death. The lungs were extremely congested.[96]

Other cases are mentioned, similar to those of Drs. Coale and Williams, in which, although the quantity taken was large, and the signs of suffering in the stomach, and the general symptoms, presented the most violent character, the traces of a corrosive poison have been comparatively insignificant.[97] The period of survivance

[94] Boston Med. and Surg. Jour. 1850, p. 279.

[95] Lancet, Dec. 1845.

[96] Wade, Lancet, June, 1848, p. 498.

[97] *Vide* Taylor on Poisons and a case by Dr. Hodges, Am. Jour. Jan. 1855.

seems to make little difference in the aspect of the alterations found post-mortem. Dr. Coale's patient lived eleven days, and the two reported by Taylor between four and five days.

Ordinarily, the change in the intestinal canal occurs as ecchymotic spots, accompanied by sanguineous effusions. The kidneys present a lively injection of the renal parenchyma,—especially near the Malpighian bodies; the epithelial cells are deformed, granular, and partially destroyed, obstructing the canaliculi. The blood is, ordinarily, black and fluid. The alteration in the kidneys, above described, would coincide with the albuminuria, which occurs in poisoning by mercury. It is rather remarkable that, in cases of poisoning from the external application of mercury, precisely the same traces of inflammation, ulceration, and gangrenous patches, are observed as in cases from its internal administration. There is a case reported by Vidal,[98] of the poisoning of a woman by the application of the acid nitrate of mercury as a lotion, instead of a liniment, to the cutaneous surface of the whole body. The internal surface of the stomach was red, the vessels were injected, and there were ecchymotic spots. The same ecchymoses were observed under the mucous surface of the bladder, and the whole length of the intestines. The blood was black and fluid. In the microscopical examination, the renal parenchyma was found very much injected,—especially in the vicinity of the Malpighian bodies; the epithelial cells were deformed, granular, and partially destroyed. M. Flandin, by chemical analysis, has recovered a sensible quantity of mercury from the tissue of the liver; the other organs showed no traces of the poison. It would seem, therefore, that, like other poisons, corrosive sublimate may cause a fatty degeneration of vital organs, thus destroying cell life and functional activity of the organs, unless death shall supervene too early (within forty-eight hours); and that similar post-mortem appearances occur after absorption through the skin, as, after its administration by the mouth, rectum, or vagina.

Mr. T. W. Bogg[99] reports a case of poisoning by corrosive sublimate, in a very puny infant, in which the post-mortem appearances were similar to those above described. He speaks, however, of the readiness with which this poison can be obtained in England, because the sheep farmers keep large amounts of corrosive sublimate on hand for use in lotions for sheep washing.

A case, reported by M. Barthélemy[100] to the Société de Médecine

[98] Gazette des Hôpitaux, Juliet, 1864.
[99] Lancet, Dec. 21, 1878.
[100] Ann. d'Hygiène, July, 1880, p. 337.

Légale de France, of poisoning by bichlorid of mercury (corrosive sublimate), shows not only that a single dose of less than 11 grains (0.70 gm.) may cause a fatal, subacute poisoning in the space of nine days, but that the most serious lesions may occur in the kidneys. The immediate symptoms in this case, following the ingestion of the poisoning, were vomiting, stomatitis, salivation (expectoration of saliva and clots), deep-seated eschars, and ulceration of the mucous membranes, abundant alvine discharges, and collapse, but no suppression of urine, though there was albuminuria on the fifth day. The autopsy showed fluid blood, absence of lesions in the liver, and a granular and fatty degeneration of the kidneys, the tubes of which were choked up with fat. A second case was reported at the same meeting of the society, where the usual post-mortem lesions were supplemented by the same congestion and fatty degeneration of the kidneys, though the structure of the liver was normal in appearance.

After symptoms of acute poisoning,—especially when the corrosive salts have been taken,—the caustic whitening of the mouth, fauces, and gullet, the edema of the glottis, the shriveling of the mucous membranes, and the profuse hemorrhages of the mucous membranes of the intestines and other parts of the digestive tract, may be especially noted; in fact, the mucous membrane, from its entrance to its exit in the rectum, may be seriously inflamed and sometimes destroyed, the latter effect being the result of the concentration of the poison. The usual congestion in the walls of the stomach with ecchymoses and partial destruction of the mucous surface is quite marked. Sometimes the walls of the stomach are very much blackened, which is probably due to the conversion of the corrosive sublimate into one of sulphid of mercury.

It should be noted that the post-mortem changes in the stomach and intestines may follow, as shown by two cases reported by Anderseck and one by Taylor, when the poison has been absorbed by the application of an ointment containing corrosive sublimate, or the solution of this salt in alcohol (80 grains to the ounce) has been applied to the skin or to the scalp.

While we have described post-mortem appearances following the ingestion of corrosive sublimate, it should be remarked that the same appearances will follow poisoning by the other salts of mercury, nitrate, cyanid, iodid, sulphid (turpeth mineral), and the white precipitate.

209. Elimination of mercury.— Toxicologists emphasize the great importance of mentioning the paths through which mercury passes

out of the system. We have already mentioned, in our opening chapter, the importance of this consideration as bearing upon medico-legal questions. While mercury may be present in the system, and remain there some time without showing any physiological action, it may certainly—especially if iodid of potash has been administered after the salts of mercury have been taken—and suddenly manifest its poisonous action, even when the patient may not have taken any further mercurial medication. Tuson has administered to a horse, first, 4 grains (0.26 gm.), then 5 grains (0.32 gm.) of corrosive sublimate twice a day for a period of fourteen days. On the discontinuance of this medication, no mercury could be detected in the urine of the horse; but, at the end of three weeks, it was found. Voit,[1,2] several years ago, presented the theory that the mercury formed a combination with albumin, which would retard its poisonous action until, by a process of oxidation, it would become separated and present its active, poisonous effects. Chemical experiments for the purpose of establishing the channels of elimination of mercury absorbed into the animal tissues show that its main outlet is through the kidneys, while the mercurial compounds of small solubility pass out of the system mainly with the feces and in the form of mercuric sulphid; this is 95 per cent of the total amount recovered by chemical analysis. It should also be noted that of 87 doses of calomel administered to a hound during a period of 31 days, about 94 per cent was recovered by chemical analysis,—from the feces 2.1175 grams, from the urine 0.0550 grams, and from the other tissues 0.0344 grams. The total amount given was 2.789 grams during the period. Mr. Blyth, in commenting upon the above experiments, justly remarks: "When, as not unfrequently happens, the mercurial poison has entered by the skin, it is evident that the most likely localities are the urine, the liver, and the kidneys." When these facts of elimination are considered, it may explain why, in certain recorded cases of mercurial poisoning, mentioned by some toxicologists as showing no evidence of the presence of the mercury, but detected in the tissues by chemical reaction, the poison might have been found if the contents of the whole of the intestinal canal had been subjected to analysis.

It should be remembered that traces of mercury may be discovered in many dead bodies, on account of the many uses of mercury, which may be handled during life, and remain in the system in small quan-

1-2 Physiol. chem. et de Unters. Augsburg, 1857.

tities. The conclusions of all chemical research for mercury should be qualified by this general statement, originally referred to by Taylor in his Medical Jurisprudence.[3]

210. Chemical examination, in general.— We take the following lucid exposition of the chemical analysis for corrosive sublimate from Dr. Guy's work on Medical Jurisprudence.

"Tests.—We may have to examine the poison in the solid form, in solution, and in organic liquids.

"Corrosive sublimate in the solid form.—On the supposition that we are ignorant of the nature of the substance submitted to analysis, we first heat a small quantity on platinum foil. It is completely volatilized. It may therefore be arsenic, corrosive sublimate, or calomel. The great solubility of corrosive sublimate in water distinguishes it at once from the other two substances. The addition of a few drops of liquor potassæ places the nature of the substance beyond a doubt. Corrosive sublimate is changed to a yellow color, while arsenic undergoes no change, and calomel is turned black. We may obtain still further assurance by the following tests: 1. Hydrosulphuret of ammonia changes the powder to a black color. 2. A solution of iodid of potassium turns it to a bright scarlet. 3. Moisten a clean rag with dilute muriatic acid (1 part of the acid to 2 of water), sprinkle upon it a small quantity of the powder, and rub it on a clean plate of copper. A silvery stain is formed, which is readily volatilized by heat. 4. Mix 1 part of the poison with 3 or 4 parts of calcined carbonate of sodium; place the mixture in a reduction tube, and apply the heat of a spirit lamp, having previously dried the upper part of the tube. A ring of globules will be formed on the cool sides of the tube.

"Corrosive sublimate in solution in water. Sulphureted hydrogen. —On the supposition that we are ignorant of the contents of a liquid submitted to analysis, we test for a base by sulphureted hydrogen. Corrosive sublimate is one of those which yields a black precipitate, first giving a milk-white appearance to the liquid. With liquor ammoniæ it gives, in common with lead and bismuth, a white precipitate; but with liquor potassii, a yellow (the hydrated peroxid). By this we recognize a persalt of mercury. The supernatant liquor contains chlorid of potassium; and, if we add to it nitrate of silver, we obtain the white chlorid of silver, which proves that the salt of mercury is a chlorid. This precipitate, being collected, washed, and dried, and heated in a reduction tube with carbonate of sodium, gives a well-defined ring of mercury."

[3] Vol. I. p. 288.

This precipitate is also insoluble in hot, dilute nitric acid, which enables us readily to distinguish it from the other black precipitates produced by the same reagent, such as lead, bismuth, copper, and silver; it is, however, readily soluble in nitromuriatic acid (aqua regia), which reconverts it into the form of corrosive sublimate.

The following are additional tests:—

"Protochlorid of tin.—A solution of this substance throws down a white precipitate, turning rapidly to gray, and from gray to black. This consists of minutely divided mercury, from which the supernatant liquor may be decanted, or separated by filtration. On introducing into the tube containing this precipitate a plug of blotting paper, and pressing it firmly against the bottom of the tube, the globules are made to coalesce, so as to form a drop of mercury.

"Metallic test (Reinsch's test).—Acidulate the liquid with a few drops of muriatic acid, and introduce a narrow slip of clean copper. A gray film will be formed on the surface of the metal. This, being carefully dried, may be introduced into a reduction tube, and heated with the flame of a spirit lamp. A ring of metallic globules will be deposited on the upper part of the tube. Pure tin, zinc, or silver may be substituted for copper. The latter is to be preferred to any other metal."

If a few, small fragments of iodin be placed between two watch glasses with this tube, and very gently heated, the iodin will unite with the mercury to form mercuric iodid, which can be easily recognized by its color. This is an extremely delicate test.

"Galvanic test.—Take a narrow strip of sheet zinc of a size convenient for introduction into a reduction tube; moisten it, and take up as much gold leaf as will adhere to it. Introduce this into the solution, slightly acidulated with muriatic acid; the gold will soon be covered with a gray film. Remove it from the solution, and dry it carefully in the heated air, above the flame of a spirit lamp. Introduce the dried metal into a reduction tube, and apply the flame of a spirit lamp. A ring of metallic globules will be formed. This test is one of extreme delicacy, and will give a characteristic result, when all other tests fail. It is that which should be preferred for the discovery of very minute quantities of the poison. The metallic deposit may be readily obtained by placing a drop of the acidulated solution on a surface of clean copper or gold, and touching the moistened metal with a fragment of zinc or iron. Dr. Wollaston once employed a key and a sovereign for this purpose. The acid in combination with the mercury may be shown to be the hydrochloric, by testing the fluid

from which the mercury has, by any of the foregoing methods, been precipitated; on the addition of the nitrate of silver, we obtain a white precipitate, the chlorid of silver, which is insoluble in nitric acid.

"Corrosive sublimate in organic liquids.—As the poison is very soluble, it is rare to meet with it in a solid form. But when it has been taken in mass, it may sometimes be separated, by merely stirring the liquid, at the same time adding, if it be very viscid, distilled water. The corrosive sublimate, from its great weight, will subside, and may be collected. As the poison is decomposed by the secretions of the body, by the mucous membrane, and by several articles of food, it might not be found in solution in the stomach, even though no antidote had been given. We must, therefore, expect to find it in one of two states,—in solution, or in combination with the solid contents of the stomach. In the former case we procure a clear liquid by diluting with distilled water, boiling if necessary, and filtering. In the latter case, one of two processes may be adopted. We may boil the solid matters in distilled water, and in this way bring the soluble salt of mercury into solution; or, if the solid matters treated in this way yield no trace of mercury, in consequence of the soluble salt having been decomposed, evaporate to dryness, and digest the dried residue in warm nitromuriatic acid. The insoluble compound of mercury is thus reconverted into the soluble bichlorid. This acid liquor must be evaporated to dryness, and the residue be dissolved in distilled water, and filtered." The corrosive sublimate may now either be dissolved out by ether, or at once tested by the protochlorid of tin, or by the galvanic, test.

Corrosive sublimate can be distinguished from calomel by its solubility in water, alcohol, and ether, by its reactions with sodium or potassium hydrates (yellow precipitate), with potassium iodid (salmon-colored or scarlet precipitate), and with ammonia water (a precipitate, white with corrosive sublimate, and black with calomel).

211. Isolation from organic mixtures.— Corrosive sublimate, and all of the other compounds of mercury, as well as metallic mercury itself, can be detected in organic liquids and tissues, as far as their mercurial nature is concerned, by the same process as was recommended in the case of arsenic (see *supra,* § 178). After removing the organic sulphur compounds, and any sulphid of arsenic or antimony which may be present, by dissolving them with the mixture of ammonia water and ammonium sulphid, the sulphid of mercury will be left as a solid, black precipitate upon the filter, since it is insoluble in the ammonia solutions. This black precipitate should then be treated with hot,

dilute nitric acid, which will dissolve any sulphid of lead, copper, silver, or bismuth if present, but leave the sulphid of mercury, which may then be dissolved by nitromuriatic acid (aqua regia), the solution evaporated to dryness, the residue dissolved in water, and this solution, which will be one of corrosive sublimate, may be tested by any or all of the above tests.

In legal cases a globule of mercury obtained by the protochlorid of tin test, by Reinsch's test, or by heating in a tube with sodium carbonate, should always be taken into court as one of the *corpora delicti,* since its fluid and metallic nature can always be recognized by the jury.

Where corrosive sublimate has undoubtedly been the cause of death, it has not always been found in the body of the deceased. Thus, in a case reported by Dr. Wegeler, of a young man who poisoned himself with 3 drams of this substance, and died on the sixth day thereafter, none of the poison could be detected in the stomach or intestines.[4] In another, by Dr. Taylor, where 2 drams were swallowed, and the man died in four days, no mercury was detected in the stomach or tissues.[5]

Orfila (the nephew of the distinguished toxicologist) undertook numerous experiments for the purpose of ascertaining what length of time was required for the disappearance of certain poisons from the system. With respect to corrosive sublimate, he states that, when it has been administered for some time, it will generally disappear from the organs in eight or ten days, and he found it but once on the eighteenth day after its discontinuance. An individual had undergone a course of treatment with corrosive sublimate, and died four months after ceasing the course. He was poisoned with a mercurial preparation. On analysis, mercury was found in his organs. Hence, according to this author, the mercury could not have been derived from the preparations taken four months before death. He also says that, if a man survives fifteen days after being poisoned with corrosive sublimate, it is quite probable that the experts will find no trace of mercury. They will, however, commit a gross error, if they conclude from this that there has been no poisoning.[6]

Any mercurial preparation may induce analogous symptoms to those which have been described under the head of corrosive sublimate as acute, subacute, or chronic poisoning. The corrosive effects

[4] Canstatt's Jahresbericht für 1846, Bd. V. p. 81.

[5] Op. cit. p. 377.

[6] Am. Jour. Med. Sci. from Comptes Rendus, Jan. 15, 1852.

upon the mouth and gullet may, it is true, be absent, but the ulceration, salivation, and inflammatory action upon the primæ viæ are usually present. By some physiologists, principally Prof. Cl. Bernard, it has been assumed that the mercury is changed by the secretions into corrosive sublimate, but the truth of this theory has not yet been substantially proved.

212. Chemical examination for nitrate of mercury, and other salts of mercury.— This depends chiefly upon the detection of the mercury, which may be accomplished in the same manner and by the same tests as described above in connection with corrosive sublimate. Mercuric nitrate also responds to the tests for nitric acid or nitrates (see *supra,* § 106), and the sulphates respond to the tests for sulphuric acid or sulphates (see *supra,* § 87). The oxids, sulphids, white precipitate, and calomel, are all insoluble in water or alcohol. The sulphocyanid of mercury also responds to the tests for sulphocyanic acid, the principal one of which is the formation of the blood-red solution, when treated with a solution of sesquichlorid of iron, similar to that caused by the same reagent with a solution of an acetate or meconic acid, a constituent of opium; but readily distinguished from the former by the fact that it is not bleached by a slight excess of hydrochloric acid, and from the latter, by the fact that it is bleached by corrosive sublimate and nascent hydrogen, while the meconate of iron is not bleached by either of these.

The separation of all of these compounds from organic tissues and fluids, so far as the mercury is concerned, is by the same method as described above under corrosive sublimate (see *supra,* § 211).[7]

XIV. Barium, salts of.

213. In general.—The chlorid, carbonate, nitrate, sulphid, and acetate are all white compounds. The chlorid, nitrate, and acetate are crystalline; the carbonate and sulphid, amorphous. All except the carbonate are soluble in water, with neutral reaction. The carbonate is soluble in dilute HCl with the liberation of CO_2.

The preparations of barium which have proved fatal are the chlorid, carbonate, nitrate, sulphid, and the acetate. Mr. Blyth reports 26 cases of fatal poisoning which he has collected up to 1894. In 15 of these cases, 9, or 60 per cent, proved fatal, and 3 were suicidal; in 3 cases, barium chlorid was taken for Glauber's salt (sodium sulphate); in 1, for Carlsbad's salts; in another, a mixture

[7] Page 279.

of barium nitrate and sulphur, for pure sulphur; in the sixth case, a combination of barium acetate and raspberry syrup, for sodic ethylsulphate; in the seventh, a larger dose put up by the apothecary than ordered by the prescriber; and in 4 cases, barium carbonate was mixed with flour and made into pastry. Kobert reports a total of 25 fatal cases of poisoning by barium salts; in Vienna, 17 in ten years.

214. Chlorid of barium.—A student of medicine took three teaspoonfuls of this salt by mistake for sulphate of magnesium. He was seized with tormina and vomiting, his extremities became cold, pulse irregular and feeble, and his hands and feet paralyzed. He recovered gradually in three days.[8] Two cases are referred to by Dr. Taylor, in which it proved fatal.[9] A healthy young woman took less than a teaspoonful of chlorid of barium, mistaking it for Epsom salts. In half an hour she had burning pains in the stomach and bowels, with vomiting and purging, followed by the symptoms of collapse above described, and a scarcely perceptible pulse; there ensued, also, great impairment of muscular power, labored respiration with bronchial effusion, coma, convulsions, and death, nineteen hours after the poison had been taken. Sensibility did not seem to be impaired.[10] This salt seems to have a decided action in some cases upon the brain and heart, producing vertigo, headache, deafness, and convulsions.

215. Carbonate of barium.— This salt is also said to have destroyed life in two cases.[11] In a case observed by Dr. Wilson, of London, the quantity taken was half a teacupful, but emetics were given, and operated before any symptoms showed themselves. In two hours the patient complained of dimness of sight, double vision, headache, tinnitus, and cramps, with occasional vomiting and purging the next day. Recovery, however, took place.

216. Symptoms of poisoning.— Kobert, who also says that even neutral salts are poisonous, separates the symptoms of poisoning by barium as distinguished between its local action, and that of the absorbed salt. He divided these symptoms into the following six groups: —

(1) Local, consisting in malaise, nausea, salivation, vomiting, and pain in the stomach. This group merges so much into the next, as hardly to admit of precise separation.

(2) Excitation of the alimentary canal, both of the nervous and muscular apparatus. Hence, vomiting, painful colic, and acute diar-

[8] Am. Jour. of Med. Sci. Jan. 1852, From Casper's Wochenschrift.

[9] Taylor on Poisons.

[10] J. Walsh, Lancet, Feb. 1859, p. 211.

[11] Parke's Chemical Essays, II. 219.

rhea. All these phenomena may be produced in animals by subcutaneous injection, and, therefore, do not depend alone upon local action.

(3) Excitation of the brain motor centers, which leads to convulsions, and may result in paralysis. About half the recorded cases of barium poisoning in man have had convulsions; the other half, paralysis. In one case there was mania.

(4) Muscular contractions, weakened or destroyed; the muscular chest movement, recorded graphically on poisoned frogs, shows a veratrin-like convulsion curve; in man, the effect shows muscular weakness.

(5) Its influence on the heart and blood vessels shows precordial distress, slowing of the pulse, strong cardiac pulsation (not only self-experienced in the patient, but heard and felt by the bystander. Arteries incompressible and rigid, blood pressure elevated. Old persons with weak blood vessels are liable from this pressure to have pulmonary hemorrhages, blood from the stomach and other organs. In frogs the heart stops in systole.

(6) Conjunctivitis and catarrh; also irritation of the mucous lining of air passages, including the nose.

Soluble barium salts, if poisonous, generally destroy life within the first three days; if the patient survives this period, recovery generally takes place (Kobert). Attention of the reader is directed to the case of barium poisoning in the Appendix, No. XXXI.

217. Lethal dose.— There is great uncertainty in regard to the satisfactory naming of the least fatal dose of barium salts. A dose of 100 grains (6.5 gm.) of barium chlorid has proved fatal to an adult woman in fifteen hours; one of 4 drams (16 gm.) of barium nitrate has been fatal to a man in six and a half hours; barium carbonate in the dose of 60 grains (3.8 gm.) has also been followed by fatal poisoning. Pirondi and Lisfranc have gradually increased the administration of the dose of barium chlorid, given daily in divided doses, from 4 decigrams up to 3 grams (46 gr.), without producing poisonous symptoms. The former himself took, in one day, 119 grains (7.7 gm.) without bad effects. This occurred in 1836. Kobert states the lethal dose as 3 to 15 grams. Dr. Tidy[12] reports a case of a man suffering from a case of rheumatism, but without other abnormal conditions, who took a mixture of barium nitrate, flowers of sulphur, and potassic chlorate, in the place of sulphur. The symptoms developed were blisters on the tongue, a burning pain in the gullet and stomach, with vomiting, diarrhea, convulsions, aphonia, and coldness of the ex-

[12] Pharm. Jour. June, 1868.

tremities. It is not unlikely that this mixture, when received into the stomach, and meeting with free hydrochloric acid in the stomach, may have, in Dr. Tidy's case given above, produced a sulphid of barium, the action of which is more violent than that of the other barium salts. The chlorin may also have been set free from its association with potassium, and increased the irritant action upon the œsophagus and stomach.

218. Treatment.— The correct treatment in poisoning from the soluble barium salts is sulphate of magnesium or sodium sulphate, in solution, pumped into the stomach and out again, because the barium sulphate is insoluble. The subcutaneous injection of atropin is suggested for increasing the action of the heart and relaxing the tension upon the blood vessels. The recovery progresses slowly.

219. Action.— In addition to the action, as interpreted from the symptoms as described by Kobert in the six groups named above, later investigators, Gmelin, Onsum, Cyon, and Böhm (1824–1874), have rather held to the theory that the salts, named as soluble, poisonous agents, do not act in their original formation; but, on being received into the stomach, the hydrochloric and organic acids contained in the stomach form other soluble compounds. Onsum goes still further and forms the theory, judging from the fact that the right side of the heart was full of blood from backward engorgement, and from discovering on post-mortem examinations that the small arteries were plugged with small fibrinous coagula with an inorganic nucleus, that the barium salts circulate in the blood, in insoluble composition, and are arrested in the lungs, thus causing minute emboli, much in the same way that a finely divided solid would act when introduced directly into the jugular vein. On the other hand, Cyon experimented by introducing barium chlorid into the jugular vein of a rabbit, and, into the other veins, sodic sulphate. He found no coagula in the small arteries, and the capillaries in the lungs were clear. Böhm, experimenting on frogs, likened the action of small doses of barium salts to cicutoxin, 1 to 2 centigrams, subcutaneously injected into frogs, acting like a heart poisoning. Again, Kobert mentions the fact that the renal tubules contain granular masses which form crystals with sulphuric acid.

220. Diagnosis.— A combination of retardation of the pulse, increased arterial tension, muscular spasms, succeeded by muscular weakness, vomiting of masses containing baryta.

221. Post-mortem appearances.— In one of the cases of poisoning by chlorid of barium before referred to, the stomach presented a uni-

form, red appearance, with clots of blood and bloody mucus scattered over it; near the cardiac end was a perforation about half an inch in diameter within, and half as wide outside, the edges swollen, and the mucous coat thickened. The small intestines also exhibited signs of inflammation. Without doubt, as is remarked by Dr. Christison, the perforation was, in this instance, an accidental occurrence, not due to the chlorid of barium. In a case of poisoning by the carbonate of barium, reported by Dr. M. Seidel,[13] of Jena, in a healthy, pregnant woman twenty-eight years of age, death took place thirty-four hours after the ingestion of the poison. The stomach mucous membrane contained numerous ecchymoses, both in large patches and in spots, and between the ecchymoses it was pale and covered with mucus; ecchymoses were also seen beneath the outer, serous covering of the stomach, and under the mucous membrane of the duodenum for a space of 8 inches (21 c. c.) from the stomach. The liver was somewhat enlarged, free from blood, brittle, and of yellowish-red color. The kidneys were congested. The mixture taken was a compound of sugar and carbonate of barium, which had been put up for a rat poison. The stomach was found to contain 0.124 gram (about 2 gr.) of carbonate of barium, and traces of barium were found in the liver.

222. Chemical examination.— The soluble salts of barium, when introduced into a colorless alcohol or gas flame, impart a green color to it; and this flame, when examined with a spectroscope, gives the peculiar barium spectrum. They are also all precipitated from neutral or alkaline solutions by ammonium carbonate, in the form of a white, granular precipitate, carbonate of barium; this is readily soluble in acetic or hydrochloric acid with effervescence. They are also all precipitated by sulphuric acid, or a soluble sulphate, in the form of white sulphate of barium, which is insoluble in nitric or hydrochloric acid. The sulphate of barium thus formed can be decomposed by fusing it with sodium carbonate and charcoal, so as to form a sulphid of barium which is soluble. It is also precipitated from a solution acidulated with acetic acid, by a solution of potassium chromate, in the form of a yellow barium chromate, care being taken that no free mineral acid be present.

223. Fallacies.— In separating barium from organic mixtures, we must bear in mind that the sulphates, normally present in the animal fluids and tissues, will partially, if not entirely, convert the soluble barium salt, which may have been taken during life, into the insolu-

[13] Eulenberg's Vierteljahrsschrift f. ger. Medicin, 1877, N. F. XXVII. p. 213.

ble sulphate; and also that in the destruction of organic matter for the detection of the mineral poisons, by hydrochloric acid and potassium chlorate, the method commonly used, the organic sulphur compounds, such as albumin, are oxidized, and sulphuric acid is one of the products of the oxidation, so that the soluble barium compounds, taken during life, may be entirely converted into the insoluble sulphate, and cannot be detected in the filtrate, after treatment with hydrochloric acid and potassium chlorate, but must be sought for in the undissolved mass remaining upon the filter by drying it, igniting in a platinum evaporating dish, moistening with nitric acid, and igniting again. The residue should then be mixed with four times its amount of mixed carbonates of sodium and potassium, and fused until the whole mass becomes liquid; after cooling, the fused mass should be extracted with water, filtered, and washed; the residue remaining will consist of carbonate of barium, which can be dissolved in hydrochloric acid and tested by the above tests. If, by chance, any barium is not converted into sulphate, but remains dissolved in the form of chlorid after destroying the organic matter with hydrochloric acid and potassium chlorate, it can be detected and separated by the methods commonly used in qualitative analysis.

XV. Potassium chlorate.

224. In general.— Since the publication of the last edition of this work, chlorate of potash has been recognized by writers on toxicology as a poison. The information, obtained from the careless use of this salt in solution, as also in its dry state, for the treatment of local sore throat and for diphtheria, brought about several accidents from unquestionably larger doses than required for successful treatment of these affections. One case was published in the Appendix to our previous edition, to which we refer our readers, as an illustration of a very strong solution of about 10 drams (39 gm.) in two glasses of hot water. Ordinarily, 6 parts of this salt will dissolve in 100 parts of water at 15° C. (59° F.); at 100° C., 60 parts of the salt will dissolve in 100 parts of water. Peterson and Haines mentioned a case reported by Potter,[14] in which two teaspoonfuls were taken in two days for a sore throat, the symptoms being violent, intestinal irritation, with black stools, considerable urinary disturbance with black urine, great prostration, and evidences of grave alteration of the blood. Witthaus and Becker have collected 89 cases of poisoning from

[14] Med. Rec. 1895.

chlorate of potash, 76 of which were fatal; almost all were accidental; 6 were supposed to be suicidal, and 3 homicidal. Ignatieff[15] reports a fatal case, also referred to by Peterson and Haines, in a man aged thirty-six years. After taking a teaspoonful of chlorate of potash, he developed cyanosis, renal pain, violent vomiting, jaundice, and bloody urine. He died on the evening of the third day. Mr. Blyth has collected from medical literature 39 cases of poisoning by chlorate of potash, 28 of which ended fatally.

225. Symptoms of poisoning.— These are mainly those of great depression; feeble pulse; cyanosis (blue, slaty complexion); sometimes diarrhea, greenish-colored; weakness of the heart; difficulty of breathing; sometimes vomiting, jaundice, and bloody urine. When absorbed into the circulation, it has a peculiar, destructive action on the red corpuscles of the blood, converting the hemoglobin into methemoglobin, and causing the secondary symptoms due to this condition, that is, hemoglobinuria, suppression of urine, the discharge of bloody casts in the urine, delirium, coma, and death resulting from the inflammation of the kidneys.

226. Lethal dose.— For a child three years old, 46 grains (3 gm.), and an adult four times that dose. When a large dose is taken in divided doses, the poisonous effects are even more serious, on account of the continued action upon the renal system.

227. Treatment.—Immediately after the swallowing of the poison, the stomach pump, using a large quantity of water, should be used to empty the stomach, and repeated to prevent further absorption of the poisonous agent. Death may occur from acute poisoning, within six hours to two days.

228. Action.— The original object of administering chlorate of potash in solution in cases of sore throat and diphtheria was the knowledge of the facility with which this agent parts with its oxygen by the aid of heat. Marchand,[16] in experimenting with the effect of chlorate of potash on animals, found, on mixing chlorate of potash or chlorate of soda (potassic chlorate or sodic chlorate) with fresh blood, the clear, red color, which first appeared, passed within a few hours into a dark, red-brown color, which gradually became pure brown. This change was produced by a 1 per cent solution, in from fifteen to sixteen hours; with a 4 per cent solution at 15° C. every trace of oxyhemoglobin disappeared within four hours. The blood soon became

[15] La Méd. Moderne, Paris, 1894.
[16] Virchow's Archiv. f. path. Anat. Bd. 77, Hft. 3, § 455, 1879.

syrupy, and, with a 2 to 4 per cent solution of the salt, became jelly-form. This jelly had much permanence and resisted putrefactive changes for a long time.

Marchand fed a dog weighing 17 kilos, with 5 grams of chlorate of potash for a week. No symptoms appearing, the dose was doubled for two days; there still being no symptoms, he gave 50 grams of chlorate of soda in five doses. On the following night the dog died. The blood was found, after death, to have a sepia-brown color, and remained so when exposed to the air. The organs, generally, had unnatural, brown color; the spleen was enormously enlarged; the kidneys were swollen, having, on section, a dark chocolate-brown color (almost black-brown), the color being nearly equal both in the renal substance and in its capsule. On examination by the microscope, the kidney showed its canaliculi filled with brownish cylinders, casts consisting of altered blood. Spectroscope showed weak, hemoglobin bands and a narrow band in the red. Diluting the blood, the hemoglobin bands disappeared, but the band in the red remained. The diluted blood, when exposed to the light, still retained its coffee-brown color.

A second experiment of a hound, a little less than half the weight of the preceding, was tried in the following manner: Doses of 3 to 5 grams of chlorate of potash were given during sixteen hours, and the animal was killed by being bled to death seven or eight hours after last dose. The same effects were produced as in the first experiment, kidneys being intensely congested, and the coffee-brown color particularly noticed. These experiments on animals correspond entirely to the action upon man, in whom the main symptoms are those of nephritis, the fatal dose varying with the condition of health of these organs; half the fatal dose, given in a case of existing nephritis, may produce fatal results. Dr. Fountain, in 1858, in experimenting upon himself, took 8.7 drams (33.8 gm.) of chlorate of potash; he died on the seventh day from nephritis. A man, 30 years of age, died in four days after taking 12.3 drams (48 gm.) of sodic chlorate in six hours.

229. **Elimination.**— Chlorate of potash is not only quickly absorbed by the mucous membrane, but also through the broken skin, and is rapidly separated from the body by means of the renal system. Isambert, with Hirne,[16a] in 1875, made some quantitative estimations, and recovered from the urine about 95 per cent of the amount of this

[16a] Gazette Méd. de Paris, Nos. 17, 35, 41, 43.

salt which had been taken by the mouth. Hehner made several experiments upon himself, taking 2.5 drams (about 9 gm.), and detected, in an hour and a half afterwards, the chlorate of potash in the urine; at that time 17.23 per cent of the salt had been excreted, and at the end of eleven hours, 93.8 per cent was recovered.

The especial action of chlorate of potash upon the coloring matter and corpuscles of the blood appears to be undoubted. The hemoglobin is destroyed in part and the corpuscles dissolve. These destroyed physical properties of the blood pass by the circulation through the kidneys, spleen, etc., and probably block up the tubules of the kidneys; thus explaining the curious coloring seen in these organs after death, and the inflammatory condition of the kidneys.

230. Post-mortem appearances.— The mucous membrane of the stomach is often found swollen, soft, and easily detachable. Extravasations of blood may be noticed in the submucous tissues. The blood presents a chocolate color, and the kidneys show evident mark of acute nephritis. The spleen is swollen, and has the same characteristic color of the blood as is shown in the kidneys.

231. Chemical examination.— The chlorate of potash crystallizes in colorless, rhombic tables, which have no water of crystallization. It is readily soluble in water (16.5 parts at 15° C.), and only slightly soluble in alcohol. Upon being heated, it gives off oxygen gas, a residue of chlorid of potassium remaining. Warmed with hydrochloric acid, chlorin gas is evolved. Heated upon charcoal, deflagration takes place. When rubbed up with many organic substances, such as sulphur or sugar, in a mortar, explosion takes place, which often results in injury to the manipulator. It can be separated from organic mixtures in the same manner as the nitrate of potassium.

XVI. Bismuth.

232. Properties.— Bismuth subnitrate is a white, amorphous, odorless, tasteless powder, insoluble in water or alcohol, soluble in hot, concentrated nitric acid, and precipitated from this solution on dilution with water.

233. Occurrence.— In medicine, bismuth is most commonly used in three forms,—the citrate, subcarbonate, and subnitrate. The last is by far the most common, and is the only one that need be specially mentioned, as the general chemical properties of all three are about the same, as well as the action.

Formerly, salts of bismuth used to be commonly contaminated by

arsenic; and several fatal cases, at first reported as being due to poisoning by bismuth, were, on further investigation, proved to be due to the impurity of arsenic.

234. **Preparations.**— (U. S. P.) Bismuth citrate, bismuth and ammonium citrate, bismuth subcarbonate, bismuth subnitrate.

The two cases of poisoning, mentioned in the early edition of this work, occurred so long ago, when bismuth was not unfrequently adulterated with arsenic, that we have excluded them in the present edition. The first case was reported in 1831, and since then there has been but one death attributed to the use of this drug. Many years ago arsenic was not an unfrequent combination with bismuth, in the form of arseniate of bismuth, owing to its incomplete preparation; and even at the present time a trace of arsenic may be found in almost all of the preparations of subnitrate or subcarbonate of bismuth, although the process recommended by the United States Pharmacopœia for the manufacture of these compounds is intended to eliminate all but the merest trace of arsenic from them. The largest amount of arsenic recorded in recent years, found by analysis of various preparations of subnitrate and subcarbonate of bismuth, is, so far as the writer has been able to learn, 1/6 of 1 per cent. The presence of the arsenic, as a contamination in these bismuth preparations, is due to the fact that the bismuth ores, from which they are manufactured, are arsenical, and it is very difficult to separate the arsenic from the bismuth, is working upon a large scale. The Pharmacopœia of 1890 prescribes a method for detecting arsenic as an impurity of bismuth; consequently, the preparation should be presumed to be free from arsenic.

Of later years there have been reported symptoms of poisoning following the application of surgical dressings and ointments containing subnitrate of bismuth, the purity of which, on chemical tests, was undoubted, and the powder found free from arsenic. One case is recorded by Mr. Blyth of fatal poisoning of an adult in nine days, who took 120 grains (7.7 gm.) of the subnitrate of bismuth; yet recovery has taken place from a single dose of two or three times this amount; there was no surety that the bismuth was free from arsenic. Experiments upon animals, however, prove unmistakably[17] that bismuth salts, whether administered subcutaneously or intravenously, cause death in from one to two days, the fatal issue being preceded by convulsions.

[17] Feder-Meyer, Rossvach's Pharmak. Dorpat, 1884; Arch. Exp. P. Bd. XX. Unters. III. 1882. No. 23; Steinfeld, 1886.
Wirkung des Wismut. Inaug. Diss.

235. Symptoms of poisoning.— The symptoms of poisoning from internal use of bismuth, as also through its absorption from surgical dressings or ointments, are the well-recognized blue stains on the edges of the gums in the mouth, stomatitis, loosening of the teeth, ulcerations of the mucous membrane of the mouth, catarrhal inflammation of the intestinal tract, and the inflammatory condition of the kidneys due to the excretion of metallic substances not normally present in the body. After large doses of bismuth, there are experienced, in cases of poisoning, a metallic taste in the mouth, pain in the throat, vomiting, purging, cold surface, and spasms of the arms and legs. It is not impossible that these last-named symptoms of poisoning may have been due to the presence in the bismuth of salts of arsenical impurities, especially as in the cases where these symptoms have been noted there is a strong garlicky odor of the breath, though this, by some, has been attributed to the impurity of tellurium. According to Kobert, the symptoms are stomatitis, black discoloration of the gums, inflammation of the tongue, albuminuria, catarrh, and inflammation mainly of the large intestine.

236. Lethal dose.— This is unknown; but in the few fatal cases recorded it is very large,—from 4 drams upwards (about 16 gm.).

237. Treatment.— Saccharated ferrous carbonate to counteract the formation of sulphohydrate in the intestines; diuretics which will produce watery discharge of urine, and eliminate the bismuth by the kidneys; use of chlorate of potash as a gargle for the mouth.

238. Action.— From the researches on animals by Meyer and Steinfeld, the action may be due to the conversion of the bismuth salts (*e.g.*, carbonate or nitrate) into a sulphid salt,—especially in the large intestine where the sulphur gases are more likely to be met; for, in their experiments, the sulphur preparation of the bismuth, given by the mouth, caused blackening of the mucous surfaces of the mouth and stomach, and was followed by ulcers, with the same effect upon the large intestine. Meyer proffers the opinion that the sulphohydrate precipitates bismuth in the tissue parenchyma, the particles of which obstruct the capillary vessels, thus producing local necrosis, while that which escapes precipitation is excreted by the kidneys. Kobert states, in addition to this action, that there is analogy between the spasms following the bismuth poisoning to those which follow lead poisoning, and also similar paralyses; he also mentions that the presence of bismuth is found in the milk of a nursing mother. Bismuth is apparently excreted by the bowels in the form of sulphid. It has also been found in the urine, spleen, liver, saliva, and the

epithelium of the mouth of persons, after taking one of its preparations.

239. Diagnosis.— By the peculiar discoloration of the gums similar to that following lead poisoning, and by the ulcerations found in the mouth and also in the large intestine, which is accompanied with albuminuria.

240. Post-mortem appearances.— State of mouth similar to that following poisoning by mercury; little, if any, inflammatory condition of the stomach and small intestine; ulceration and black stains on the inside of the large intestine; the microscope will show brownish-black granules of sulphid of bismuth in the vessels of the mucous membrane of the large intestine.

241. Detection and tests, chemical.—Bismuth is readily separated from organic mixtures by the same process recommended for the separation of arsenic and the other metals. The sulphid of bismuth is separated from the other sulphur compounds in the same way as sulphid of lead; it can then be dissolved in nitric acid, the excess of acid expelled by evaporation, and the solution of nitrate of bismuth employed for the tests for bismuth, the principal ones of which are the following: (1) Sulphurated hydrogen precipitates bismuth in the form of the black sulphid, which is insoluble in hydrochloric acid, but soluble in nitric acid. (2) Ammonia water precipitates the white hydrate of bismuth. (3) A solution of the nitrate or chlorid of bismuth, when treated with a large excess of water, becomes turbid, on account of the decomposition of the salt, the subnitrate or subchlorid, which is insoluble, being precipitated. This precipitate is insoluble in tartaric acid. (4) Carbonate of sodium precipitates the basic carbonate of bismuth, a bulky, white precipitate. (5) Chromate of potassium precipitates the yellow chromate of bismuth, which is soluble in dilute nitric acid, but insoluble in potassium hydrate, thereby differing from the chromate of lead. (6) A solid compound of bismuth, mixed with carbonate of sodium and heated on charcoal with the blowpipe, gives brittle globules of metallic bismuth, the charcoal at the same time becoming coated with oxid of bismuth, which is orange while hot, but becomes yellow when cold.

XVII. Chlorin.

242. In general.—The principal source of poisoning by this gas is its use in bleaching and laundrying establishments. There are sev-

eral cases of accidental poisoning reported, and one fatal case in which a negress lost her life. Chlorin is a yellowish-green gas, two and one half times as heavy as air, of a very irritating, penetrating odor, very soluble in water.

243. Symptoms.— Orfila has shown, by experiments upon animals, that a saturated solution of chlorin in water produces effects similar to those of the mineral acids. The inhalation of chlorin vapor induces coryza, difficult breathing, and distress, which soon pass off when the irritating vapor is not inhaled.

If it is inspired in an atmosphere containing a large proportion of the gas, as may sometimes occur in the manufacture of disinfectants and bleaching powders, and care be not taken to wear a "respirator" to prevent the pungent gas from coming in contact with the mucous membrane of the nose and air passages, accidents are liable, and have sometimes been reported, to occur. In these cases the local irritation produces a cough with hemorrhage. Prof. Holland[17a] mentions a fatal case as having occurred at Cornell University in 1894.

The chlorin vapor, coming in contact with a moist, mucous surface, is converted into hydrochloric acid, and will produce the same caustic effects as that acid; the corneal surface of the eye becomes opaque from an alteration of its anatomical structure.

Continued exposure to inspiration of an atmosphere with this irritant gas will produce bronchitis and bronchopneumonia and, later, coma with enfeebled heart action and death. In these cases there may be a purulent discharge, and sometimes a destruction of the mucous membrane of the nose.

244. Lethal dose.—Lehmann found, in animals subjected to its inhalation, that 1.5 parts in 1000 parts of air caused dyspnea and coma.

245. Post-mortem appearances.— Hyperemia of the lung tissue, patches of pneumonia and catarrhal inflammation of the bronchi; in the stomach, ecchymosis of the mucous membrane.

246. Treatment.— Exposure to pure air, the usual treatment for bronchitis, and appropriate treatment to stimulate the heart's enfeebled action.

247. Tests, chemical.— The gas can generally recognized by its odor. It bleaches moist litmus paper. Passed through a solution of silver nitrate, it forms a white precipitate of silver chlorid, which is soluble in ammonia and insoluble in nitric acid. In solution as chlorin water, it has the same action.

17a Peterson and Haines op. cit. p. 380,

XVIII. Bromin.

248. In general.— Bromin is never used medicinally. It is chiefly met with in photographers' studios and in chemical laboratories. It is a dark reddish-brown liquid (sp. gr. 2.97); volatile at ordinary temperatures; its vapor is reddish-brown, and is so heavy it can be readily poured from one vessel to another. It is somewhat soluble in water, "bromin water," quite soluble in alcohol, ether, chloroform, and carbon disulphid.

There are only two cases of poisoning by bromin which we can find recorded,—one mentioned in our former edition, another of a girl aged ten who died within seven hours. The latter was given a mixture by a quack, containing a solution of bromid of potassium, to be taken with chlorin water; she took three doses; this, on the addition of chlorin water, would set free about 2 grains of bromin.

249. Symptoms of poisoning.— Intense burning pain in the throat and stomach, with eructations of bromin vapor; this is followed by collapse; death in the few cases of fatal poisoning recorded has occurred within eight hours. The other case on record of fatal poisoning by bromin in man has been reported by Dr. Sayre, of New York.[18] A. H., aged twenty-four, of good health and temperate habits, a daguerreotypist by profession, residing at Williamsburg, near New York, swallowed 1 ounce (30 gm.), by weight, of bromin, for the purpose of destroying himself. The immediate symptoms, as reported by his medical attendants, were spasmodic action of the muscles of the pharynx and larynx, and great difficulty of respiration. This was soon followed by intense burning heat in the stomach, with great anxiety, restlessness, and trembling of the hands. The pulse was rapid, tense, and corded, and the respiration greatly hurried. The stomach was entirely empty at the time of the taking of the bromin. Various means were used, unsuccessfully, for his relief. The symptoms above described increased in intensity; the hands and feet became cold, with failure of the pulse, etc., until 2 P. M., when he died, seven and a half hours after taking the poison.

250. Subacute poisoning.— A few reported cases of its absorption by handling, and the inhalation of its vapor in laboratories and photographic workshops, have shown that 6 centigrams per kilo weight have proved fatal. The symptoms of poisoning were shown by yellowish coloration of the skin, formation of blisters, difficult respiration, bronchitis, nasal catarrh, conjunctivitis, keratitis, spasm of

[18] New York Jour. Med. Nov. 1850.

glottis, dizziness, headache, and numbness. When taken internally from unwashed hands there was swelling of the mouth, vomiting of yellowish-brown masses, etc.

251. Lethal dose.— As named above in the few cases reported, 6 centigrams per kilo body weight.

252. Treatment.— Inhalation of aqueous vapor, and the administration internally of milk, alkalies, for instance, soda, albumen, and mild aqueous diuretics.

253. Action.— Is caused by substitution of bromin products formed with the albuminous substances and epithelia. These, coming in contact with moist, mucous surfaces form hydrobromic acid, and are then afterwards changed into bromin albuminates. Both bromin and hydrobromic acid, producing local irritation or inflammation of the respiratory mucous surfaces, may cause bronchitis, bronchopneumonia, etc. In the stomach, if the mucous surface is protected by food, the action of the bromin will be exerted upon the food, as well as upon the mucous surface. The absorption of the bromin albuminates produces narcosis and stupor, from the action upon the nerve centers.

254. Diagnosis.— Characteristic odor of free bromin, yellow discolorations, and, on dissection, the vapor of bromin is freely given off from the tissues of the dead body.

255. Post-mortem appearances.—After inhalation of bromin vapor there is frothy mucus in the air passages, ecchymotic spots in the lungs, and evidence of pneumonia, with brown discoloration. After ingestion of large doses, the mucous lining is brown and tough like tan; there are erosions, gangrene, and perforation of the stomach, with signs of inflammatory action of the kidneys. The coagulation of the blood is quite extensive. The surface of the body shows the skin as if tanned and partly necrosed.

The following is a detailed description of Dr. Sayre's case, the symptoms of which have been above related: The post-mortem examination was made seventeen hours after death. On opening the abdomen, the external surface of the stomach was found vividly injected, as was also the peritoneal coat of the duodenum, and of the mesentery. A portion of the latter nearest the stomach was stained a deep yellow color, as were also other parts lying immediately beneath the stomach. A softened, ecchymosed spot, 1½ inches in diameter, and several others of a smaller size, were also found upon the peritoneal coat of the stomach. The stomach contained about 4 ounces of thick fluid, resembling port wine dregs, and exhaling faintly the odor of bromin. Its whole internal surface was covered with a thick, black

layer, resembling coarse, tanned leather. The mucous membrane was very thin, and there was intense submucous injection.

256. Detection.—Free bromin may be recognized by its odor, color, appearance, the brownish color of its solution in chloroform or carbon disulphid, and forming with phenol a crystalline mass of tribromphenol, insoluble in water.

257. Bromid of ammonium, of calcium, of ethyl, of lithium, of potassium, of sodium, and of zinc, in general.—The bromids, *viz.*, of ammonium, of calcium, of ethyl, of lithium, of potassium, of sodium, and of zinc, deserve mention in the light of poisons. Potassium bromid (bromid of potassium) presents a more intense form than the action of the other bromids, including that of hydrobromic acid. Acute fatal poisoning from the bromids alone is unknown.

258. Special acute and chronic symptoms.—The most decided effects are those which follow a dose continuously repeated. Among these may be mentioned the diminished reflex sensibility of the mucous surfaces, which is due to the local action of this salt upon the mucous linings of the passages communicating with the outside air; also, a slow and feeble pulse, loss of voice, peculiar, disagreeable odor (fetid) of the breath, congestion of the fauces, a hoarse cough, tottering gait, muscular weakness,—especially of the legs; loss of intellectual activities, rather a mental hebetude, then unconsciousness; finally hallucinations and mild delirium. The evidence that death follows these long-continued doses of bromid of potassium, disassociated with other drugs, is very unsettled. The effects of chronic medication by potassium bromid simulate the general paralysis, not uncommon where this medicine is administered, and there is confusion between the symptoms of the disease and those caused by the treatment by the drug. The acne above noticed is sometimes associated with a profuse erythema and cutaneous ulcers, which persist during the conditions of the administration of the drug. The long-continued dosing with the bromids is called "bromism."

259. Treatment.— For the treatment of these skin diseases, the administration of tincture of cinchona and small doses of Fowler's solution of arsenic has the reputation of preventing the liability.

260. Action.— Experiments on animals and man, by the writer[19] and by others, seem to show that the absorption of bromid of potassium has an effect of reducing the caliber of the smaller blood ves-

[19] Amory on Action of Bromids of Potassium and Ammonium, Boston; also, monograph by E. H. Clarke and Amory, published by James Campbell, Boston, 1872. Comm. Mass. Med. Soc. Vol. XV. pp. 606–623.

sels (capillaries), and thus renders the tissues anemic and incapable of performing their functions. The action of this salt is apparently not dependent upon this combination with potassium, the action of this latter salt being primarily upon the heart, as mentioned in describing the action of the neutral potassium salts; for the amount of any potassium salt required to act on the heart is larger than that which is contained in the ordinary doses in the compound of bromid of potassium. The medicinal dose of potassium bromid, given in solution, is from 8 to 128 grains (½ to 8 gm.) two or three times a day.

XIX. Iodin.

261. Occurrence.—Iodin is used in solution as the tincture and as Lugol's solution. The tincture is an 8 per cent solution, and Lugol's solution contains 5 per cent iodin and 10 per cent potassium iodid. Iodin is also used widely in medicine in the form of salts called "iodids" and in organic combination as iodoform. In nature, iodin is found in all vegetable or plant life in the sea.

262. Properties.—Iodin crystallizes in scales, bluish-gray in color, and having a metallic luster (sp. gr. 4.95). Volatile at all temperatures, and if heated, gives off copious violet-colored vapors. Only slightly soluble in water, it dissolves readily in hydriodic acid and in solutions of iodids, especially potassium iodid. It is readily soluble in alcohol, ether, chloroform, and carbon disulphid, forming deep violet-colored solutions.

263. Preparations.— (U. S. P.) Compound solution of iodin; tincture of iodin; iodin ointment. (Unofficinal) Iodin paraffin; iodin glycerin; iodin alkalies; iodoform.

Of the 15 cases reported in Mr. Blyth's table of fatal poisoning, 2 were suicidal, 13 from accident or negligence. The accidents which result in poisoning by preparations of iodin are usually due to mistaking this for some other medicine, or by its injection into abscess sacs, or by external application to wounds.

264. Symptoms of poisoning.— This substance is capable of acting in a deleterious manner upon the system, under two circumstances, *viz.*, by the long-continued use of small doses, or by the administration at once of a large quantity. Acute poisoning is caused, usually either by taking into the stomach large quantities of tincture of iodin, or of Lugol's solution (solution of iodin in iodid of potassium, "Liquor iodi compositus," U. S. P.), or by injecting solutions of the above preparations of iodin into internal cavities, such as ovarian

or other cysts. But few of the former class of cases are recorded which are either suicidal or accidental. Dr. Bainbridge[20] records one case of accidental poisoning, due to the ingestion of 1 ounce of tincture of iodin, representing ½ dram of iodin, by mistake for the compound infusion of senna. Dr. Gairdner[21] records a fatal case in a child four years of age, caused by 20 grains of iodin in the form of tincture; and Van Hasselt and Husemann report two suicidal cases by tincture of iodin. A large number of fatal cases due to the injection of tincture of iodin into ovarian cysts have been reported. Velpeau has collected records of thirty fatal cases out of one hundred and thirty operations.[22]

The symptoms occasioned by taking an excessive dose of iodin into the stomach are those of irritation or inflammation of the stomach and intestines, such as are produced by other irritant poisons, pain in throat, gullet, stomach, and abdomen, with vomiting, or, perhaps, only nausea. Vomitus is usually blue from the action of the iodin on the starchy matters, and has the peculiar odor of iodin. There is also usually diarrhea, the stools being colored with iodin and often with blood. Urine is generally suppressed. In the fatal cases due to the injection of iodin solutions into ovarian cysts, the general symptoms are similar to those caused by the taking of such solutions into the stomach. The symptoms occasioned by too prolonged use are incessant vomiting and purging, pain in the abdomen, heat and dryness of the throat, headache, rapid emaciation, violent cramps, and a general febrile condition. A patient of Zink, a Swiss physician, after taking too large medical doses of iodin for about a month, became restless, had burning heat of skin, tremors, palpitation, very frequent pulse rate, violent priapism, copious diarrhea, excessive thirst, emaciation, and occasional syncope. He died after an illness of six weeks. Salivation is also an occasional result of the prolonged use of iodin. A case is related in which one dram of the tincture of iodin, in about an ounce of spirit, is said to have proved fatal.[22a] Very often, however, large doses of this substance are productive of no evil effects, nor are unpleasant results generally experienced from its prolonged administration. Dr. Christison quotes a case in which a child three years old, took 3 drams (11.66 gms.) of the tincture, and suffered only from thirst and slight vomiting.

Bellot[22b] reports a case of attempted suicide of a woman who drank

[20] Medical Times and Gazette, Dec. 28, 1861, p. 669.

[21] Essay, 1824, p. 20.

[22] Husemann's Handbuch der Toxicologie, Supplementband, p. 131.

[22a] Prov. Jour. June, 1847, p. 356.

[22b] La Méd. Moderne, Paris, 1893.

a half glass of the tincture. A quart of water was given, but there was no vomiting for an hour; the vomit was a dark, thick fluid, afterwards followed by clear blood; prompt treatment by milk and starch did not prevent pains in the belly, tenderness, dizziness, and syncope; she, however, recovered in a few days. The urine was normal, and no iodin could be detected either in the urine, saliva, or sweat. It would seem from this that most of the poison was ejected from the stomach without being generally absorbed into the system.

265. Lethal dose.— It is difficult to state what the minimum lethal dose would be; probably the iodin would form iodid of potassium from the potassium compounds in the digestive tract; this would account for the variability in the appearance of poisonous symptoms following large single doses of iodin, as well as when it is removed from the stomach by appropriate and prompt treatment.

266. Treatment.— Washing of the stomach with sodium hyposulphite, and its removal by stomach pump; internal use of sodium carbonate, sulphanilic acid, sodium sulphanilate, ice, opiates; if necessary, tracheotomy; the use of starch in solution, and its removal with the stomach pump.

267. Action.—The action of iodin upon the animal tissues would be, first, to form a compound of iodin albuminates, which will rapidly further decompose. Iodin, being less likely, at the ordinary temperature of air, to form vapor than chlorin or bromin, is not likely to be inhaled in large enough quantities to cause more than local action upon the upper part of the air passages, and its poisoning by inhalation rarely occurs, if at all. In contact with the mucous membrane and the skin, iodin produces inflammation. When absorbed, iodin, probably as iodid of potash, produces an irritation upon the tubules of the kidney, causing albuminuria and hemoglobinuria. Its action upon the mucous membrane of the throat and air passages will cause bronchitis, difficult breathing, asthma, and a peculiar, nasal catarrh.

268. Diagnosis.— The peculiar, nasal catarrh caused by "iodism" is recognized by an eruption upon the mucous membrane of the upper air passages. The odor of iodin is characteristic. It may also be recognized in the urine by the starch tests producing a blue color.

269. Post-mortem appearances.—These are, in brief, stomatitis; tumefaction of the stomach and duodenum, with yellow-brown coloration; hemorrhages; pseudomembranes in the pharynx and larynx. There may be fatty degeneration of the heart, liver, kidneys, and muscles, and inflammation of the tubules of the kidneys. In chronic

cases, acne or ulceration of the skin and also of the intestinal tract is usual.

In the case related by Zink, redness of the intestines, in some places approaching to gangrenous discoloration, was the chief morbid alteration observed. Interesting, in connection with poisoning by injection into ovarian cysts, is the observation of Edm. Rose,[23] that free iodin could be detected in the stomach and intestines.

270. Detection and tests.— The most delicate test for free iodin is the addition of a few drops of a starch solution, when the mixture will become deep blue, due to the formation of an iodid of starch. This blue color will not appear if the free iodin is first combined with an alkaline hydrate into an iodid salt. The blue color will disappear on gentle warming, and reappear on cooling. If the solution is heated too high, the blue color will not reappear, as the iodin has been volatilized. If the iodin, instead of being in a free state, is combined with a base, such as in potassium iodid, it must first be set free by the adding of a little chlorin. On adding a solution of silver nitrate to a solution containing iodin, free or as an organic iodid, a faintly yellowish precipitate is formed, insoluble in nitric acid and nearly so in ammonic hydrate.

XX. Lead and salts of lead.

271. Occurrence.—The chief sources of lead compounds that have been used for homicidal cases, and hence are of forensic interest, are the acetate or subacetate.

Other salts, such as the carbonate and chromate have often given rise to cases of poisoning both acute and chronic, but rarely have they been fatal.

The normal acetate, often called sugar of lead, is used extensively in the arts, and the carbonate of lead, white lead, is very largely used in painting. The chromate and the oxids are also largely used as pigments.

272. Properties.— Lead acetate is a white, crystalline solid, which effloresces in the air, soluble in water and alcohol, has a sweetish taste at first, hence its name, afterwards becoming metallic, and an acid reaction.

Lead subacetate, or basic lead acetate, is formed when a solution of lead acetate is boiled with an excess of lead oxid. It is soluble in

[23] Virchow's Archiv. XXXV. 1866, p. 12.

water, and is present in the liquor plumbi subacetates, or Goulard's extract.

Lead carbonate is insoluble in water. It forms the base of many oil paints known as white lead paints, which are a mixture of this salt of lead and the hydrate. It is the salt which is the most common source of chronic lead poisoning.

Lead chromate and oxid are both insoluble in water, soluble in dilute, vegetable acids and in hydrochloric acid; hence, by the latter they may be a source of chronic lead poisoning.

273. Preparations.—(U. S. P.) Lead acetate; lead carbonate; lead carbonate ointment; lead iodid; lead iodid ointment; lead nitrate; lead oxid; lead oxid plaster; subacetate; cerate (Goulard's cerate) and solution of sugar of lead; white lead. (Unofficinal) hair dyes; litharge, lime, and starch; litharge, lime, and potassic bicarbonate.

274. Deaths from lead poisoning.—Though there have been a large number of deaths from lead poisoning both on the Continent and in Gt. Britain (and in the city of New York for twenty years, 113 cases), yet but few of these are homicidal or suicidal. Most of the reported deaths from lead poisoning are from accident, either by mistaking the lead for some other substance, or by working in lead factories or paint shops. The cases in which this drug has been used with criminal intent are very rare, and in each case the acetate of lead was used mixed with food or drink. One case[24] occurred in France, and two[25] in England. It is more often by accident or for suicidal purposes that mixtures containing lead are the cause of death. The officinal, dilute solution of subacetate of lead, containing three per cent of the subacetate, commonly called Goulard's lead-water, has thus accidentally been mistaken for water; or, in one recorded case, when used as a wash, it has been sucked off of sore nipples by a child, and with fatal consequences, as death occurred after eleven days.[26] Death has been caused by drinking a solution of lead salt, from mistaking a bottle of lead wash for a cathartic. It has also caused serious consequences where used to color children's toys. Finally, death has occasionally followed the medicinal use of lead, when used to counteract diarrhea or the spitting of blood in phthisis, but this always in case of enfeebled persons.[27]

275. Sources of lead poisoning.—The principal sources of acute lead poisoning are the two acetates of lead, sugar of lead, and the basic

[24] Ponchou, in 1842.
[25] Central Criminal Court, 1844, and Chelus Ford, summer assizes, 1847.
[26] Taylor, op. cit.
[27] Fouquier, Leridon, and Ruva.

acetate of lead (Goulard's extract), and the carbonate of lead or white lead. Chromate of lead (chrome yellow) has also caused acute poisoning, but will be referred to below under a separate heading, since the chromic acid of the compound appears to be an important factor in producing the symptoms. The numerous sources of chronic lead poisoning will be enumerated in speaking of chronic lead poisoning (see *infra*, § 278).

The metallic lead is poisonous to animal life, no matter in what form it may be swallowed; the rapidity in the appearance of the symptoms, however, depends upon the form in which it may be administered, because some of the salts of lead are more easily absorbed into the system than others. All the compounds of lead may, therefore, cause acute poisoning, though Dupasquier at one time denied this fact. More recent experience shows that even the insoluble salts and metallic lead itself can, after ingestion, cause poisoning, the rapidity and intensity being in proportion to the degree of solubility in the secretions of the body; for instance, chlorid of lead combined with common salt, causing the most rapid, poisonous consequences.

When it is remembered how varied are the means for the employment of lead in the industrial pursuits, it may not be surprising to find that chronic lead poisoning is very common. There are a large number of employments which may be the source of the slow absorption of lead, and consequently give rise to chronic lead poisoning. The following extract from an article contributed by the writer[28] will show how universal is the danger of the absorption of small amounts of lead, and how difficult it is sometimes to trace the poisoning to its source:

"The subject of chronic lead poisoning is becoming more and more important every year, on account of the gradually increasing use of lead in the arts, which augments the number of sources of poisoning, and renders the detection of the source in some cases exceedingly difficult. . . . A lawsuit has recently occurred, in which a white lead company has been sued by a workman, who contracted the disease while in the company's employ.

"Of the numerous sources of chronic lead poisoning, by far the most important, and those giving rise to the greatest number of cases, are the use of lead carbonate (white lead) in painting, and the use of lead or lead-containing pipe for the conduction of drinking water.

[28] The Sanitary Engineer, N. Y. Aug. 3, 1882, p. 185.

"With regard to the use of white lead as a pigment, the greatest danger arises during the process of its manufacture, and the utmost care should be taken by the workmen to prevent the inhalation or ingestion of the lead dust, those portions of the process, in which the dust is disseminated throughout the air of the workroom, such as the grinding and packing, being the most dangerous. With constant care and watchfulness on the part of the workman, little fear need be apprehended of poisoning, and a new workman should always be informed of the danger attending the employment, and instructed with regard to the precautions to be taken to prevent poisoning. These precautions are chiefly the following:

"1. The use of respirators in all workrooms which are liable to be dusty. Any lead-containing dust coming in contact with the mucous membrane of the mouth, nose, or throat is naturally swallowed, dissolved in the gastric juice, and absorbed. Swallowing is not, however, necessary to absorption, since the lead compound can be absorbed from any surface, even through the unbroken skin, although it is extremely slow in this way and may be prevented by strict attention to cleanliness.

"2. No article of food or drink should be brought into a workroom. All food and drink should be taken where there is no possibility of its being contaminated with lead dust. Before partaking of any drink, the workman should thoroughly rinse out his mouth and throat, and should wash his moustache, if he has one.

"3. The workman should not eat any food until he has thoroughly washed himself and changed his clothing. In some European manufactories the workmen are obliged to change all of their clothing, before either entering or leaving the workrooms, suitable anterooms being provided for the purpose, and to thoroughly wash themselves before putting on their clean clothing.

"4. Strict attention to cleanliness. All lead-containing dust should be removed from the skin upon leaving the workrooms, so as to prevent absorption through the skin, which takes place upon long contact. Special attention should be paid to the hands, face, and head, no dust being allowed to remain in the hair or beard.

"In some factories sulphuric acid lemonade is provided for the workmen to drink. This may help to a certain extent in preventing or delaying the absorption of those lead compounds which are swallowed, by forming with them lead sulphate, which is insoluble in water and acids, but it does not entirely prevent it, because lead sulphate is slightly soluble in the intestinal fluids. In one factory

I have known of milk being provided for the workmen to drink, and, it was said, with excellent results in preventing lead poisoning.

"With regard to painters, many of whom are affected with lead disease (to such an extent that the lead colic has received the name of painter's colic), the principal precaution necessary is cleanliness, since they are exposed to danger arising from white lead dust only while mixing the paint; but the same care should be taken as to cleanliness as in the case of white lead manufacturers.

"The use of lead pipe for conducting drinking water is very extensive, and yet comparatively few cases of poisoning result from it. This is due chiefly to the fact that certain hard waters cause a deposit of an insoluble coating, consisting of lead sulphate and carbonate, upon the inner surface of the pipe, which prevents any further action of water upon it; and that the people, generally, have learned the necessity of thoroughly emptying from the pipe the water which has stood in it for several hours. All waters do not act upon lead pipe with equal readiness. As a rule, the purer the water, the more readily does it attack metallic lead. Therefore soft waters take up the most lead and hard waters the least. Hard waters, however, vary in this respect. If a hard water contains only lime and magnesian sulphates and carbonates without any alkaline sulphates or carbonates, it has no action upon the pipe except to deposit the insoluble coating upon its inner surface; if, however, alkaline salts are also present, the alkaline carbonate takes up a little of the lead salt, however hard the water may be.

"Lead cisterns should never be used for containing drinking water, nor, as a rule, should lead pipe be allowed to project beneath the surface of the water in a well, although in the case of most hard waters no serious consequences would result. It is better to use pure block tin pipe beneath the surface of the water, soldering it on to the lead pipe above the highest level of the water in the well. Then, only the small amount of water which is contained within the pipe has an opportunity to attack the lead; and if the pipe is thoroughly emptied of the water which has stood in it for several hours, no serious consequences will result. Tin-lined lead pipe and tinned lead pipe are, in my opinion, more dangerous than pure lead pipe, since the galvanic action, set up by contact of the two metals, increases very much the action of the water on the lead.

"Although the above-mentioned are the two principal sources of chronic lead poisoning, they are by no means the only ones; these sources are so numerous, that, in certain cases, the physician finds it

very difficult, or even impossible, to trace the poison to its source. Any employment in which metallic lead or any compound of lead is used, is liable to cause chronic lead poisoning. Among the more common employments may be mentioned working in lead mines, or any mines in which plumbiferous ore is handled; working with metallic lead, as in the case of plumbers and typesetters; the handling of lead compounds, as in the case of glazers of porcelain and pottery ware, manufacturers of enamel, etc.; the use of lead chromate (chrome yellow) and red lead (minium) as pigments; the use of lead or any of its compounds in any cooking utensil, or article used for preserving food,—especially acid fruits and vegetables, which are often put up in soldered tin cans. The acid contained in some of these—as tomatoes—may easily remove a dangerous amount of lead from the solder. The use of lead-containing enamel for lining ironware to be used for cooking is very dangerous. I have personally known of several cases of acute and subacute lead poisoning resulting from cooking tomatoes in such enameled ironware, and one fatal case in an infant, due to drinking milk which had been warmed in one of these vessels. This particular vessel is in my possession, and yields to water, to which a few drops of acetic acid have been added, when allowed to stand a few hours at the ordinary temperature, an amount of lead which might produce symptoms of poisoning in an adult. The addition of lead compounds to articles of food or drink; thus, many yellow candies are colored with lead chromate, and many olive-green ones with a mixture of lead chromate and Prussian blue. Snuff has been known to be colored with lead chromate and with red lead. A not uncommon source of chronic lead poisoning is the cleaning of glass bottles with shot. The greatest danger arises in the case of champagne bottles, one or more shot sticking in the angle between the side and bottom of the bottle, owing to the carelessness of the bottle washer; the carbonic or other acid of the wine, malt liquor, or cider, readily removes a dangerous amount of lead from the shot. I have in mind a recent case of lead colic, due to drinking bottled ale, the lead being derived from shot left in the bottles. Wine is sometimes sweetened by treating it with oxid of lead (litharge) or sugar of lead. The use of lead pigments for coloring children's playthings is very dangerous, the child being liable to put the toy in its mouth and suck off some of the poison. The use of spurious (lead-containing) tinfoil is another source of chronic lead poisoning; some of this foil consists chiefly of lead (one specimen examined contained about 98 per cent of lead), and is used for wrapping tobacco, pressed meat,

and other substances. There is one case of poisoning recorded of a clergyman, who was in the habit of using cachous for a sore throat, between one and two boxes being consumed daily; finally, after diligent search for the cause of the poisoning, it was found that several milligrams of lead were contained in the foil surrounding the cachous in a single box. The habitual use of some hair dyes is also a frequent source of lead poisoning, many of these compounds consisting of sulphur, sugar of lead, and some fluid, as glycerin, water, etc. Some cosmetics also contain a lead compound.

"The above enumeration shows how varied are the sources of chronic lead disease, and how difficult it often is to detect them. A case illustrating this is that of a newspaper editor, who was in the habit of sticking his clippings upon paper with red wafers which were colored with red lead, the wafers being moistened with the tongue. A limited epidemic of lead poisoning in Paris was finally traced to a baker's oven, which was heated by burning painted wood, the lead oxid, volatilized by the high temperature, was deposited on the bottom of the oven, where it came in contact with the bread."

When lead has been found in a suspected case of poisoning, it is of course important to show that the peculiar occupation or profession of the individual would not explain the presence of this metal in his organs. The experiments of the younger Orfila show that eight months is the shortest time of the sojourn of the lead in the liver, the intestines, and bones.

276. Acute poisoning.— Acute poisoning by lead has a different character from the chronic form. After swallowing the lead poison, there is perceived a sweetish taste, on the part of the victim, then an astringent or metallic after-taste; soon after, there is often a sensation of burning, distress, more or less distinct, in the throat, and extending down the gullet even to the stomach; at other times, there is simply a sensation of weight at the pit of the stomach. The tongue, often slightly swollen, is generally white, and is covered with small, prominent points. Nausea and vomiting may then occur, at first of a limpid and clear liquid which contains lead. Later on, the vomited matter contains mucus, and little white patches due to the salts of lead. The chromate of lead alone colors the vomited matter yellow. Then, very acute gastrointestinal colicky pains occur, accompanied by a contraction of the intestinal tube, and sometimes by meteorism, a tympanitic resonance of the abdomen. These pains are often associated with constipation of the bowels, though sometimes there is a diarrhea, in which the stools appear black from the com-

bination of lead with the sulphureted hydrogen gas of the intestines, producing sulphid of lead. Along with these active symptoms the limbs feel heavy, and there is a pallor of the countenance with livid lips.

The bluish-black line along the margin of the gums may not be present in acute poisoning, and may be absent in chronic lead poisoning, but this subject will be more thoroughly discussed when we treat of chronic saturnism. If the poisoning is severe, the respiration becomes stertorous and asphyxia is imminent, accompanied with a painful hiccough. Vertigo then comes on, trismus (local lockjaw), and epileptiform convulsions may supervene, preceding a comatose condition (due to a true inflammation of the brain), which is followed by death in two or three days. The intellectual faculties may occasionally remain intact up to the last moment of life.

Death from acute poisoning is uncommon, but the symptoms may be very severe. Acetate of lead in solution, commonly called sugar of lead, is followed in a very few minutes by the appearance of symptoms of poisoning such as above described. Vomiting usually occurs within the first half hour, but may be delayed from one to two hours. Often the vomited matters contain streaks of blood. The lead colic pains are usually relieved by pressure upon the abdomen. When diarrhea occurs, it is due to the irritation of the large intestine, and the presence of the sulphid of lead into which the acetate of lead is probably converted. The poisonous action is very soon developed into the symptoms, associated with the various nervous phenomena, such as headache, shooting pains in the limbs, muscular cramps in the legs, and local numbness. All the above symptoms are never present in any one case, but most usually the vomiting and the colic are present and very persistent. Usually death occurs about the second or third day. Even when recovery takes place, the convalescence may be much retarded, and the symptoms of lead poisoning may persist during a lapse of a year,—especially that of pain in the abdomen and sickness. There was an outbreak of lead poisoning, which occurred among some soldiers at Tione, in the southern Tyrol. One of the soldiers attacked died, and forty-five required treatment in the hospital. Though the symptoms of poisoning were unmistakable,—especially the effect on the mouth and the muscular weakness of the limbs,—in some, tetanic spasms, yet in none was there colic, though the constipation was very marked, and in two of the cases there was strangury.

An eminent toxicologist presents in brief the symptoms of acute

saturnism. It results from the ingestion of a soluble salt of lead, or of some drink with which it has been mixed, accidentally, in considerable quantity, such as cider, wine, or vinegar. Very soon after, nausea, not always accompanied with vomiting, supervenes, followed by very acute pains in the abdomen; sometimes there is diarrhea, but more often obstinate constipation. The countenance becomes pale, and, oftentimes from the moment of attack, there may be noticed a bluish line upon the borders of the gums. The voice becomes indistinct, a painful hiccough supervenes; syncope and terrible convulsions precede a state of comatose stupor, which may endure for two or three days and terminate in death. (Tardieu.)

The only diseases likely to be confounded with the acute poisoning are simple enteric or gastric fever. The lead poisoning can be distinguished from these by the much more acute pains in the abdomen, the state of the mouth, the line of the gums, though it may frequently be absent, the fetor of the breath, the serious nervous disturbances,—especially their rapid progress,—the tendency to constipation, and the relief of the pain by free catharsis, and often by firm pressure applied to the abdomen.

277. **Subacute poisoning.**— According to Dr. Taylor,[29] if the patient recovers from the first symptoms of lead poisoning, the secondary effects often last a considerable time. The acute poisoning, unless recovered from, usually ends fatally within a week or a fortnight; if the symptoms are prolonged beyond this period, the form of poisoning assumes that of a subacute character. In the subacute form of poisoning, when the lead has been absorbed into the tissues, its presence seems to deteriorate their nutrition and functions; hence we see disturbances in the nervous sensations, muscular motions, and in the abdomen severe colicky pains and deficient alvine evacuations. The patient becomes pale, emaciated, and feeble in body, with a general feeling of lassitude, heaviness of limbs, but without mental hebetude. The presence of lead in the systemic circulation appears to destroy the red blood corpuscles, causing the sallow pallor of the countenance, and paleness of the mucous membranes; later on, the metallic lead is deposited in the skin, the external covering of the eyes, and the mucous membranes, causing a dusky countenance, dullness of the eyeballs, and pallor of the mucous surfaces generally. This appearance is illustrated in the case of those whose occupation necessitates the handling of the metal, or respiring an atmosphere in which various forms of lead are used in manufactures or in the arts,

[29] Op. cit.

such as painting, plumbing, typesetting and the like. In plumbing, the heating of the lead evolves fumes of the oxid, often in ill-ventilated apartments, and thus exposes the workman to the dangers of acute, subacute, or chronic lead poisoning. That the same individual may practise his art or labor for a long time without showing symptoms of poisoning, and finally, at an unexpected time, be overpowered by the lead is not strange, since, either he may eliminate the poison so rapidly by proper attention to the action of the bowels, or by being fortunate enough to escape the risk of the absorption of a poisonous amount by working in the open air, or by cleanliness of person. One instance is known to the writer where a plumber, of several years' experience in his trade, was found dead in a water tank he had been lining with lead, and with sufficient evidence that his death was caused by lead absorption. His health had previously been excellent. We cite, among cases reported, the following[30] instance of poisoning from the use of lead in the arts. There is one part of the process of pottery manufacture, where the articles are briskly rubbed together causing the presence of lead particles in the air of the factory. A workman of sixteen years of age, working in one of these factories, was admitted to the Royal Infirmary at Glasgow with symptoms of acute lead poisoning, marked by emaciation, anemia, and an exhausted appearance, wrist drop, and wasting of the muscles of both arms, the contractile power of the extensor muscles of both arms being absent; his gums presented the blue line peculiar to lead poisoning; the abdomen was quite natural in appearance, soft and elastic, and pressure caused no pain. His pupils were dilated, though the sight was not much disturbed. The pulse was rather weak, and 70 to the minute. His urine had a specific gravity of 1.012, neutral, of pale color, without sediment and without albumin; the phosphates appeared on heating, but otherwise the physical examination yielded normal results.

The patient was given the usual treatment of iodid of potassium, which was, however, discontinued after three days, the bromid of potassium being substituted. On the morning of the ninth day after admission he had coma, and, in a few moments, exhibited clonic convulsions extending over the whole body; he was unconscious, with dilated pupils, foamed at the mouth, had retraction of the abdominal walls, involuntary micturition, with a pale, clammy face, and rapid and forcible action of the heart. The convulsions soon ceased, but he remained unconscious in a comatose state. The next day his tem-

[30] Lancet, Feb. 26, 1881.

perature became 102°, and on the tenth day his urine became albuminous, but no lead could be found in it.

We may have, in a case of subacute poisoning by lead, either a predominance of the symptoms referable to the nervous centers, or of those referable to a disease of the kidneys; since, like many other poisons which are eliminated by the kidneys, the effort of elimination may cause structural decay of these organs. It should be observed here that, where there is suspicion of lead poisoning, cathartic treatment, not by salines except Epsom salts, since these favor lead absorption, helps to hurry the discharge of the metal and its salts through the intestinal canal; and that when the salts of lead are used as astringents in diarrhea, unless this diarrhea be checked, absorption in the intestinal canal is prevented, owing to the rapid escape of the lead with the fecal discharge. Dr. Headland[31] speaks of the danger of using in medicinal preparations a combination either of opium and acetate of lead, on account of the chemical decomposition, or of alum and lead, on account of the increased astringency and risk of lead absorption from its retardation within the intestinal tract. Either of these agents combined with lead would naturally tend to detain the metallic salt in the intestinal canal.

The differential diagnosis between the subacute and the chronic lead poisoning is not an easy matter, since liability to both forms may occur to the same individual, and the difference depends upon the amount of the poison absorbed within a given time. This will be evident from the very nature of the case, since it may take a longer exposure in one case than in another to receive a given dose. So, also, a person in active health and of a full, vigorous habit of body, by which we mean a normal blood supply and an active nutrition of tissues, may resist the absorption for a long time, the emunctory organs discharging a large amount of lead. This reasoning will explain why, as is stated further on in this article, the presence of lead in the urine is absent in the last stages of saturnine intoxication, the kidneys in this condition being unable to perform their function of elimination, not only of lead, but of other deleterious substances from the blood and tissues.

278. **Chronic poisoning; action.**—Chronic poisoning by lead, or saturnine intoxication, or saturnism, as it is variously called, may follow exposure to the ingestion of any salt of lead, or exposure to the action of the metal itself by ingestion, by handling, or by means of inhala-

[31] On the Action of Medicines, Phila. 1868, p. 299.

tion, as has been before mentioned. E. Gaucher,[32] in an elaborate article on lead poisoning, sums up the results of his experience in the following conclusions: 1. Nutrition, in lead intoxication, is considerably retarded, as shown by a diminution in the solids of the urine, its specific gravity being diminished, the excretion of urea (which is the measure of changes caused by the metamorphosis of tissue), and that of the chlorids and phosphates being below the normal standard. 2. During the active period of this intoxication, or, in other words, of the presence of lead in the system, a large number of the red blood globules or corpuscles are destroyed, which results in a saturnine icterus, or the presence of bile in the blood (ictère hémaphéique), and the abundant discharge of the coloring matters of the blood with the urine. This deglobulation of blood, and the retardation of nutrition of the tissues, explain the early appearance of anemia in those poisoned by lead. 3. The urinary secretion presents two distinct phases, whose characteristics are opposed to each other,—in the commencement of the lead poisoning, the urine is deficient in quantity, is concentrated, and has a high color, while at the end of a certain time after poisoning, it becomes more abundant and of a lighter shade. 4. Most generally, permanent albuminuria (presence of albumin in the urine from the organic changes of the kidneys) is not so common as a transitory albuminuria. This is considered by M. Gaucher as symptomatic of lead poisoning. This toxic albuminuria is the result of a deteriorated nutrition, or destruction of anatomical elements; while the albuminuria from organic disease of the kidneys (nephritis) derives its albumin from the blood plasma. 5. The elimination of substances not natural to organic life, and which are usually and readily absorbed, such as medicaments, is retarded. This elimination goes on in an irregular way, at one time more active than at another (*saccadé*).

279. Symptoms.—With this introductory, though, perhaps, a rather more physiological synopsis of the theory of lead poisoning than most of our readers would relish, we pass directly to the history of symptoms, which may be more easily comprehended. We have spoken in this article, as well as in that on mercury, of a peculiar but rather constant appearance of a blue line along the gums. This is a very important evidence of lead poisoning, more especially so in the subacute or chronic form, since at one time it was considered pathognomonic of this metal; yet it may not always be present,—especially in that class of persons who are of cleanly habits, and brush their teeth

[32] Revue de Médecine, 1881, p. 900.

often, though the line may not be easily gotten rid of even by this means. The presence of the line is most generally a positive, physical symptom of poisoning by lead, bismuth, or mercury. Its absence is merely a negative evidence of small value, when taken in connection with the other evidences, especially the chemical evidence. The presence of this line once ascertained, the cause of it, whether mercury or lead, can perhaps be determined by analysis of the saliva and urine.

This slate-colored line, sometimes black when intense, is of about the width of 2 or 3 millimeters (about 1/10 of an inch), and runs along at the junction of the gums with the teeth, particularly of the incisors and lower canines. It is due to the deposition of the sulphid of lead, which is formed by the contact of lead with the vapor of sulphureted hydrogen, which is so often present in the cavity of the mouth, whether caused by the decomposition of particles of food lodging between the teeth, as suggested by Tanquerel,[33] or of the alkaline sulphates contained in the saliva (Schebach), or by the blood.[34] It makes no difference whether the lead is absorbed by the mouth or stomach, or by the skin or lungs; this line is formed, no matter how the poison is introduced into the system, and, in fact, it can often be made to appear, when absent in a case of lead poisoning, by giving the patient a course of treatment by iodid of potassium, and it will then persist for several weeks. Frank Smith[35] has proved the existence of this secondary or indirect line under the use of iodid of potassium, and Hilton Fagge[36] has since confirmed this statement. In the case of Schroenbröd, in 1873,[37] this line was not present until after the patient had ceased for four weeks from using a vinegar containing lead, which had brought on lead colic.

In addition to this blue line on the gums, there may be observed, in cases of lead poisoning, anatomical lesions in the mouth, which are quite constant and of a peculiar character, such as lead discolorations of the mucous membrane, and patches on the inside of the lips and cheeks before mentioned. The gums, oftentimes swollen and bloody, generally become thin and retracted, leaving the teeth prominent and encrusted with a deposit of tartar, and the teeth often undergo decay.

These peculiar appearances of lead poisoning need not be mistaken for somewhat similar ones, which have been observed with those persons who work on copper, or who use medicinally nitrate of

[33] Traité des Maladies de Plomb, Paris, 1839.

[34] Bubler. Dict. Enc. de Sci. Med. art Bouche, 1869.

[35] Lead Poisoning, Lancet, 1869, I. p. 746.

[36] Lancet, 1876, I. p. 709.

[37] Nouveau Dict. de Méd. et de Chir.

silver or iron, or who brush their teeth with a dentrifice containing charcoal, which, according to Gubler,[38] sometimes produces analogous appearances, or with those having a scorbutic exhalation. It will always be easy to distinguish the leaden line from any of the above, or even from that caused by mercury. Oxygenated water, brought in contact with the line, gives rise to a whitish trace, due to the conversion of the sulphid into the white sulphate of lead; and sulphureted hydrogen water will change the sulphate back into the black sulphid of lead,[39] and even make this line darker than it was originally. Pauvert suggests another crucial test, of scraping the incrustation off the teeth, treating it with nitric acid and distilled water, thus transforming the lead salt into a soluble nitrate; if, after evaporation to dryness and dissolving the residue in water, the liquid be shaken with a solution of iodid of potassium (1 part in 20), it will become yellow from the presence of iodid of lead.

Functional disturbances of the secretions of the mouth may accompany the presence of the above phenomena, causing salivation, inflammation of the gums, and fetid breath; the reaction of the saliva, instead of being alkaline, becomes strongly acid. These functional troubles in the mouth precede those of indigestion, with furred tongue, capricious appetite, and sometimes even nausea and distressing vomiting.

As the diseased condition advances, the tongue becomes white and dry, there is a disgust for food, and a feeling of heaviness and tenderness at the pit of the stomach. To this succeed constipation of the bowels, hardness and morbid sensibility of the whole abdomen, with pain in the back, and, frequently, headache. These symptoms are nothing more than what is often met with in aggravated dyspepsia, or in gastric catarrh; but, in lead poisoning, they culminate in a sudden attack of colicky, abdominal pain, preceded by albuminuria, which, it is needless to say, may often be overlooked. These colicky pains, feeble at first, gradually increase and become almost intolerable, being especially marked by increasing and decreasing paroxysms of pain; pressure, gently exerted over the whole abdomen, may diminish these pains, which become intolerable when the hand is suddenly removed after pressure. These attacks are in no way different from the ordinary intestinal colic caused by impacted feces, and it is not improbable that their cause may be the same. The only means of distinguishing lead colic from other colics consists in the persistency of the former and its obstinancy to the ordinary means of treatment;

[38] Op. cit.

[39] D'Arcet, Tanquerel, Grehant.

these pains may last in an interrupted manner for days or even weeks, and are usually succeeded, sometimes even accompanied, by an inflammation of the brain and an obstinate paralysis. These colics, upon the authority of Tanquerel, Kussmaul, and Maier, are attributed to lesions in the intestinal glands and muscular fibers, which may cause atrophy or destruction of both. According to Kussmaul and Maier, in the whole length of the alimentary canal, from the mucous membrane of the stomach to that of the lower portion of the colon, there is an atrophy or a fatty degeneration of the structure and anatomical elements of the glands and muscles,—especially in the intestines,—by which their caliber is narrowed, this being more apparent near the pyloric orifice of the stomach and along the small intestine. Alcoholic excesses increase the tendency to colic, and so do also the drinking of acid wines and vinegar. More recent investigations render it probable that this abdominal pain is due to a neurosis of the intestinal plexus of nerves.

It may be generally said, without going too much into detail, that lead may be deposited throughout the whole body, and wherever it is so accumulated, it will prevent the functional activity of tissue, whether it be in the blood corpuscles concerned in nutrition, in the glandular cells, which pour out secretions essential to the chemistry of life, in nerve centers, which excite and control the sensations and motions of the body, in the brain, which presides over the whole organic life, or in the kidneys, which remove deleterious substances from the circulation and the various tissues.

The constitutional effects of lead have been thus described by Tanquerel des Planches:—[40]

1. Saturnine coloration of the gums, of the buccal mucous membrane, and of the teeth. A narrow, leaden-blue, or slate-blue line, from 1/20 to 1/6 of an inch in breadth, is formed on the margins of the gums nearest to two or more teeth (usually the incisors) of either jaw. The inner part of the lips and cheeks is sometimes stained blue. The blue discoloration is supposed to depend upon the formation of the sulphid of lead.

2. Saturnine taste and breath.

3. Saturnine jaundice.

4. Emaciation, most evident in the face.

5. Slowness, smallness, and irregularity of the pulse.

The true saturnine diseases which follow may either exist alone

[40] Traité des Malad. de Plomb, Paris, 1839.

or be complicated with each other. They are: 1. Lead or painter's colic. 2. Lead rheumatism or arthralgia. 3. Lead paralysis, often accompanied with loss of sensation in the part affected. 4. Disease of the brain, encephalopathia saturnina, manifested by delirium, coma, or convulsions, and the loss of one or more senses.

The poisonous effects of acetate of lead arise gradually and become slowly developed after its long-continued use. When taken in a large quantity at once, it is capable of producing symptoms analogous to those caused by other irritant poisons. The symptoms which follow the introduction of the carbonate, or other slightly soluble salts of lead, into the body, are precisely similar to those already mentioned; occurring soon, when the dose is large, and gradually, when entering the system by water, wine, cider, or other liquids which are apt to be impregnated with them, and also when inhaled by the lungs. The subject of chronic poisoning by lead, in these ways, is one which has indeed its interest for the physician, in its relation to medical police, and the facts relating to it are fully detailed in the comprehensive works on poisons, and the treatises upon the practice of medicine.[41] The character of the poisoning differs so completely from that of the irritant poisons, that no mistake on this point can ever be made; the only embarrassment which ever presents itself being the discovery of the particular way in which the lead has been introduced into the system.

Dr. Wm. Norris, of Stourbridge, gives an account of the poisoning of a vast number of persons by acetate of lead, accidentally mixed with flour. About 30 pounds of this salt were mixed with sixty or eighty sacks of flour, which were retailed to a great many persons in the neighboring villages. Nearly a thousand persons suffered from the poisonous effects of lead. The persons who ate the bread, after a few weeks complained of a peculiar taste; some compared it to soda, others to rusty needles or copper. The tongue was covered with a darkish cream-colored mucus, and was soft and flabby; the gums were

[41] Besides the chief authorities which may be consulted on this subject are the following: Tanquerel des Planches, Traité des Maladies du Plomb (also translated by Dr. Dana, of Boston); Dr. Burton, Méd.-Chir. Trans. Vol. XX.; Gueneau de Mussy, Dub. Quart. Jour. Vol. VII. p. 405; Dalton, Am. Jour. Med. Sci. Oct. 1849; Alb. Smith, Month. Jour. March, 1853; Bois de Loury, Rev. Med. Juliet, 1852; Alderson, Lancet, July, Aug. Sept. and Oct. 1852. For some cases of poisoning by visiting cards (glazed with lead), *vide* Med. News, 1854; or Med. Times and Gazette, July 1854. Eichmann. Reynold's System of Med. London, 1876. Practice of Medicine, Tanner, Phila. 1866. Cyclopedia of Medicine, von Ziemssen, Am. ed. N. Y. 1881, Vols. VII. VIII. XI. XII. XIII. XIV. XV. XVII. XIX. Nouveau Dict. de Méd. et Chir. Paris, 1880, Vol. XXVIII.

swollen, with a blue line on the margin; in many cases the blue tinge extended nearly over the gums, occasionally on the inner side of the lower lip, and in a faint degree over the mucous membrane of the mouth and towards the fauces; the tonsils were in some cases enlarged, and in other cases there was salivation. These symptoms were accompanied by loss of appetite, nausea, vomiting, flatulency, and obstinate constipation, with a sense of constriction in the throat and epigastrium, and a violent, spasmodic pain and twisting around the navel, which was retracted. The pain was sometimes increased by pressure, and when the paroxysms were violent, the muscles of the abdomen were contracted spasmodically. A most frequent symptom was pain in the loins, about the situation of the lumbar fascia, and in the deltoid muscles. The patients were chilly, with great languor and lassitude; the skin dry; the intellect was clear, but there was general depression, and the pulse was low and feeble; the features were sallow and shrunken, and the muscles flabby; the fluid vomited was often mixed with bile and, occasionally, a coffee-ground secretion; the feces were dark and highly offensive, with scybala; the urine scanty and of a dark red color, almost like porter.[42]

An interesting case of imputed poisoning by acetate of lead may be found in Dr. Maclagan's "Contributions to Toxicology," in the Edinburgh Monthly Journal for December, 1848. Although the falsity of the charge was shown by many circumstances, which it is not here necessary to relate, there was one which, in itself, would have had great weight in its refutation. The acetate of lead was said by the prosecutor to have been given to him in coffee. Now, acetate of lead is the very agent employed to decompose and decolorize coffee, in isolating its characteristic constituent caffein. The precipitate which the lead salt forms in its infusion, if it is allowed to rest, subsides, and leaves a pale-colored fluid, in no respect resembling that which people are accustomed to drink as coffee. One ounce of ordinary coffee was boiled for ten minutes in the coffeepot, which had been used, with six cupfuls of water. It was allowed to settle for five minutes and then poured off. It had the ordinary appearance of unclarified coffee, dark brown, and slightly turbid, and depositing some coffee grounds. "Half an ounce of sugar of lead, being the same proportion to this bulk of fluid which was found in the coffee got from the prosecutor, was now added; the coffee was boiled again and allowed to settle for five minutes after removal from the fire. An-

[42] Prov. Med. and Surg. Jour. June 27, 1849.

other similar portion, being decanted, was now found to be a clear, transparent liquid, with hardly any color, except a faint shade of green, and more resembling a weak infusion of green tea than coffee. It was obvious, therefore, that if, during the breakfast, the coffeepot remained at any time at rest for five minutes, the next cupful poured off must have been so different in appearance from ordinary coffee as at once to attract attention." This decolorizing property of the sugar of lead should not, therefore, be lost sight of in any future case of alleged poisoning by its mixture with coffee.

280. Lethal dose.— It is difficult to specify the minimum fatal dose of lead, because the size of the dose required to produce poisoning varies with the individual, probably agreeing, as in other poisons, with the rapidity with which this toxic agent is absorbed and eliminated. Its elimination is by way of the intestines and kidneys, and in case of a diarrhea coexisting with the administration, the drug passes out too quickly to produce its peculiar physiological effects upon the tissues of the body. Lead salts are often used by physicians in cases of diarrhea, and though sometimes evil consequences may follow this use, the occasions are rare. A case is recorded,[43] in which a young man experienced serious consequences from taking a little more than 2 grains (13 cg.) of acetate of lead in three days, and an adult[44] died after drinking half a liter (a little over one pint) of wine out of a bottle which had been cleansed with a few grains of metallic lead. Of course the gravity of the danger and the severity of the poisoning depend upon the amount of poison absorbed, and not upon the amount swallowed. So, also, the form in which the lead is administered, as above remarked, affects the rate of absorption, as a mixture of lead and common salt is more readily absorbed than the lead itself; the subacetate of lead is more readily absorbed than the acetate. The degree of concentration also affects the size of the necessary fatal dose, since the greater the concentration, the greater will be the astringent action of the drug, and the less will be the amount absorbed at any one time. According to the experiments of Orfila, De Gaspart, and of R. Moreau,[45] acetate of lead in the dose of ½ a gram (8 gr.) injected into the veins of a dog will destroy its life in twenty-four hours, and a single dose of 10 or 18 centigrams (1½ to 3 gr.) administered in the same way will kill it in eight days.

An instance of recovery from 1½ oz. (46 gm.) of sugar of lead, swal-

[43] Nouveau Dict. de Méd. et de Chirurgie, tome XXVIII. p. 307.

[44] *Ibid.*

[45] Recherches expérimentales et cliniques sur l'Empoisonnement aigue par le plomb, Thèse de Doctorat, Paris, 1875.

lowed by mistake, is related by Dr. Taylor.[46] The woman fell ill almost directly, had a nauseous, metallic taste in her mouth, with a burning heat in it, the throat, and the stomach. On taking some water to wash away the taste, vomiting was brought on. The mouth became very dry; she had great pain at the pit of the stomach, and excessive vomiting. Two hours afterwards, she felt sleepy and stupid, alternately perspiring and shivering; she complained of violent colic, which was relieved by pressure. She had, also, cramps in the thighs, great languor, and numbness over the whole body, with giddiness. The gums were tender, and had, apparently, a blue line on their edge; there was some salivation, and the breath was foul. There were other symptoms, such as have been before detailed. She was relieved by treatment in a few days. Several other cases of the same kind, which it is needless to describe, are reported. They all recovered; as, undoubtedly, it must be admitted many cases may recover, provided a sufficient amount of the poison pass through the alimentary canal, without being absorbed into the tissues of the body.

There seems to be no agreement as to what dose is the minimum lethal dose in lead poisoning. There have been three recoveries after 1 ounce (31.1 gm.) of acetate of lead has been taken; a larger dose might be considered fatal. It should be stated, however, that, in the case of vomiting and diarrhea following the ingestion of more than an ounce of the acetate, and a little larger amount of the milder salt of carbonate of lead, an uncertain quantity of the poison swallowed may be removed, and thus leave less of the amount to be absorbed in the system, the symptoms in the latter case being less pronounced.

281. Treatment.— In acute cases of poisoning, sodium sulphate or magnesium sulphate diluted with a large amount of water should be given, and then the stomach pump used to pump out the insoluble sulphate of lead, into which the sulphate of magnesia will convert the less stable salt of acetate of lead; afterwards albuminous drinks and milk, to be followed by the administration of opium to relieve the pain, colic, and diarrhea, thus soothing the local irritation of the mucous membrane. In subacute and chronic cases, potassium iodid diluted in water should be given to form an iodid of lead in the tissues; also the use of hot baths, and electrical stimulation of the muscles; to relieve the colic, hypodermic injections of atropin, and also large doses of olive oil and opium, should be given internally.

[46] Op. cit. p. 348.

Lead salts are eliminated from the system by the urine, the perspiration, and the milk.

282. Action.— Experiments on animals would seem to show that lead will increase peristaltic action of the intestines, spasmodic muscular contractions, and produce colic, sometimes diarrhea, and death occurring with heart paralysis. The nervous symptoms predominate after the administration of several small doses of the acetate, as, for instance, with dogs from .2 to .5 of a gram. These are mainly emaciation, shivering, and paralysis of the hind legs; towards the end epileptiform convulsions. Mr. Blyth had the opportunity of studying the effects on animals, who were grazing in a pasture on which manure had been spread, which was a refuse from a plumber's yard, and contained lead as well as paint. All of the fifteen young cattle placed in this field showed, in two or three days, loss of appetite, emaciation, and diarrhea; in two, blindness supervened, the retina presenting an appearance not unlike that seen in Bright's disease; three were in a sort of delirium. Four of these animals died, and, on post-mortem, presented a granular condition of the kidneys. In other cases, to which Mr. Blyth also refers, seven cows and a bull died from eating lead paint; symptoms observed were loss of appetite, obstinate constipation, suspension of rumination, dry muffle, quick breathing, and coma. He observes that cattle have also been poisoned from eating grass which has been splashed with the spray of bullets, as in the pasture in the vicinity of rifle butts. Kobert mentions, as the action of lead poisoning, paralysis of the nervous centers, disturbed alimentation, tetanic contraction of intestines, large amount of uric acid in kidneys, atrophy of muscles. After chronic poisoning, deposit of lead in many organs; elimination by the glands, intestines, kidneys, skin, saliva, and milk.

283. Post-mortem appearances.— There are few poisons productive of so much suffering, and, when fatal, of such violent symptoms towards the close of life, and yet leave in the body such indistinct traces of their action, as these. In a case which terminated with the symptoms of saturnine encephalopathia, *viz.*, delirium, insensibility, and tetanic convulsions, Empis and Robinet found no anatomical alterations of any importance. Lead was discovered, by incineration, in the brain and liver. Likewise, in another case examined by Dr. Hopfgartner, of Vienna, lead was found in the same organs, but no pathological alterations, except that one of the lateral columns of the spinal marrow appeared to be wasted. In Dr. Letheby's case, lead was freely detected in the contents of the stomach, in the brain, mus-

cles, liver, intestines, blood, and in the serum of the cerebral ventricles. The stomach and intestines were pale and nearly empty, and the latter contracted, and in some places invaginated. Most generally a microscopical examination of the muscles will reveal a deterioration of the muscular fibers and an enlargement of their nucleoli.

In one case a post-mortem examination showed nothing particularly abnormal about the head, but the muscles of the forearm were examined microscopically, and the extensors were found to have undergone atrophy of the muscular fibers, the transverse striæ being very much less marked in these than in the flexor muscles. The paralyzed muscles had also more nuclei than in the normal extensor muscles, the fibers of the former being also very much narrower.

There is also diminished elasticity of the arterial walls, from physical alterations of the inner linings of these vessels and of their muscles; also, atrophy of the extensor muscles of the lower part of the arm, and of the nerves connecting these muscles. Contracted kidney is found filled with gritty urates; in the liver, spleen, and other similar organs there is observed the formation of vacuoles and swelling of the parenchyma, and in the brain an accumulation of serous fluid under its membrane, and under that of the spinal cord.

284. Detection and tests in general.— The detection of lead and compounds of lead is very easy, since the tests for lead are so characteristic. The principal ones are the following: (1) All solid compounds of lead, if mixed with sodium bicarbonate and heated upon charcoal with a blowpipe, are reduced, and a globule of metallic lead is left upon the charcoal; this globule can be removed from the charcoal and examined in respect to its color, malleability, hardness, etc. (2) Solutions of lead compounds, if acidulated, and sulphureted hydrogen passed through the solution, will yield a black precipitate of sulphid of lead, which precipitate is readily soluble in nitric acid, but is insoluble in hydrochloric or acetic acids. (3) If a solution of a lead salt be treated with a solution of sodium carbonate, a white precipitate (white lead) is formed, which is soluble in nitric or acetic acids with effervescence. (4) If a solution of a lead salt be treated with dilute sulphuric acid, or with a solution of a sulphate, such as sodium or magnesium sulphate, a white precipitate of sulphate of lead will be formed, insoluble in nitric or hydrochloric acids, but decomposed by hot alkalies or alkaline carbonates; hence, the value of sulphates as antidotes in cases of poisoning by lead compounds, the sulphate of magnesium being the best one for this purpose, on account of its cathartic effect, which removes the sulphates of lead from the

intestines rapidly, otherwise it would be slowly decomposed by the intestinal fluids and absorbed; this precipitate of sulphate of lead is also soluble in a solution of ammonium acetate. (5) If a solution of chromate of potassium be added to one of a lead salt, a yellow precipitate, chromate of lead, chrome yellow, will be formed which is decomposed by caustic alkalies, but is insoluble in nitric acid; this enables us to distinguish it from a similar precipitate formed by the same reagent with soluble salts of bismuth. (6) If hydrochloric acid or a chlorid be added to a solution of a lead compound, a white precipitate, chlorid of lead, is formed, which is quite soluble in boiling water, but separates again on cooling in the form of long prismatic crystals. (7) In the same way, if hydriodic acid or an iodid, like iodid of potassium, be added, a yellow precipitate, iodid of lead, is formed, which is soluble in boiling water, and separates from it, on cooling, in the form of golden-yellow hexagonal scales; free nitric acid will prevent this reaction. (8) If a piece of zinc be introduced into a solution of a salt of lead, metallic lead will be deposited upon the zinc in the form of a black granular powder, which can be melted into a metallic globule.

285. Separation from organic matter.— The method usually adopted is that used for the separation of arsenic from organic tissues, as already described in speaking of arsenic. The sulphid of lead, formed by the sulphureted hydrogen in that process, is freed from organic sulphur compounds by the mixture of ammonia water and sulphid of ammonium; the black sulphid, which is left upon the filter paper, is dissolved by nitric acid, which separates it from sulphid of mercury if present; the solution of nitrate of lead thus formed should be evaporated to dryness to expel all excess of nitric acid, and the residue dissolved in water, to which a few drops of acetic acid may be added if necessary, and this solution used for performing the above tests. If bismuth or copper compounds were present at the same time, the lead must be separated from them by precipitating it with dilute sulphuric acid, washing the sulphate of lead thus formed, decomposing it with boiling sodium carbonate, washing the lead carbonate which results, dissolving it in acetic acid, and performing the tests with this solution.

Since lead, when eliminated, passes out with the urine, although only in small amount, the analysis of this fluid for lead is very frequently necessary for the purpose of diagnosis. About a quart of the fluid should be evaporated to dryness, the residue burned in a crucible, until only a white mass remains; this complete combustion of

the organic matters of the urine is facilitated by the addition of pure niter before ignition.

This white mass is then treated with concentrated sulphuric acid till all the nitrous oxid fumes are expelled, diluted with water, and boiled for a few minutes. After it has completely cooled, strong ammonia water is added cautiously till beginning alkaline reaction, and then the whole is made acid with acetic acid. The lead sulphate is now in solution, and lead may be precipitated by passing a slow stream of hydrogen sulphid through the solution for an hour or two. The precipitate is collected on a filter, washed, dissolved in a very little, hot, dilute nitric acid, and the solution evaporated to dryness in a watch crystal on a water bath. The residue is dissolved in warm water with the addition of a drop of acetic acid if necessary, and to the clear solution a few cubic centimeters of a moderately strong solution of potassium bichromate is added, and the whole allowed to stand twelve to twenty-four hours. The lead is precipitated as the chromate, which may be collected at the center of a very small filter, carefully washed, and a stream of hydrogen sulphid passed directly against the precipitate on the filter. The lead chromate is coated with a layer of lead sulphid, and is easily seen as a brown or black deposit on the white filter after careful drying.

XXI. Salts of copper.

286. Occurrence.— Copper is widely distributed in nature, in native masses and its ores, and less abundantly, as an impurity, in many minerals and soils. It is present in minute quantities in many cereals, herbs, and drugs. It also occurs in the ash after cremation of the human body. Blyth[46a] estimates that it is ingested to the extent of about 1 milligram per diem. The metal itself cannot be considered a poison, but many of its salts act as irritants, when taken into the system in large quantities.

The most common of these salts, met with in cases of poisoning, are the sulphate (blue vitriol), the subcarbonate, and the subacetate of copper (verdigris). Arsenite of copper (Scheele's green), and the mixture of this with the acetate of copper (Schweinfurt's green), have been mentioned in connection with arsenical poisoning. Verdigris is formed in alloys of copper by the combined action of air and dampness, or by the maceration of copper in acetic acid. It has been

46a Poisons: Their Effects and Detection, London, 1895, p. 615.

made by allowing copper weights to soak in vinegar. Verdigris has been used in cooking to give to vegetables a more brilliant green color. M. Derheims has recorded a case of poisoning by liqueur d'absinthe colored with blue vitriol.

287. **Properties.**— Copper sulphate, commonly known as "blue vitriol" or "bluestone," occurs as large, deep blue crystals, very freely soluble in water, with an acid reaction and metallic taste, insoluble in alcohol.

Copper subacetate, usually called verdigris, is a mixture of copper acetate and copper oxid, generally of equal molecular proportions.

288. **Preparations.**—(U. S. P.) Sulphate of copper. (Br. P.) Nitrate of copper. Aluminate of copper, "pierre divine," in use in France and Germany as an external wash, which contains 16 parts cupric sulphate, 16 parts potassic nitrate, 16 parts alum with a little camphor. Sulphate of copper (bluestone) is the only medicinal preparation mentioned in the United States Pharmacopœia. The unofficinal acetate of copper is commonly used as an astringent injection in medicine. Verdigris is an impure acetate of copper, or a mixture of acetate and hydrate, and frequently contains impurities of chalk and sulphate of copper.

289. **Poisoning in general.**— Acute poisoning by salts of copper is not common. In Blyth's table for ten years, there were only eight deaths from this cause, three of which were suicidal and five accidental. There are no deaths returned, as caused by copper salts, during the last twenty-five years by the Massachusetts medical examiners.

The reader will find details of two cases in the Appendix to this volume, one of which is the celebrated case recorded by Maschka, the other from the New York Medical Record, in 1882. Copper is universally found in the vegetable kingdom, and is a constant constituent in our daily food, containing therein 0.95 milligram. It is contained in animal tissues in minute quantities, about 1 part in 20,000 to 100,000 parts. It is also contained in aerated water. It is also used in preserved vegetables to produce an attractive, green color, forming phyllocyanate of copper (Tschirch), which is a derivative of chlorophyl, and allied to it in composition; probably, there is also free sulphate of copper or its acetate, part of which would be dissolved by boiling in water, in the preparation of these foods for the table. The use of copper in these preserved vegetables would be an adulteration under the British pure food act. A number of convictions for this adulteration have occurred in England.

Bakers sometimes add sulphate of copper to dough, to whiten the

bread and increase its weight; especially is this the case, where an inferior or damaged flour is used, since the sulphate of copper improves the apparent quality of the bread, and helps the flour to absorb more water. Hence its more frequent use as an admixture to flour made from grain harvested after it has become too mature. In some countries, also, it is the custom to steep the seed wheat in a solution of sulphate of copper, in order to destroy parasitic insects, or to prevent the germination of the eggs of those which injure the grain after it has been sown; if the grain is allowed to steep too long in the copper solution, the seed absorbs a notable amount of copper. This risk could be avoided by using, instead of the sulphate of copper solution, one which should contain sulphate of sodium, which is equally effectual in preventing the damage threatened to the wheat, and is not injurious to animal life; for it has sometimes happened that instead of being planted, the seed has been sold and manufactured into flour, and, in consequence, serious illness has occurred to those who have eaten the bread. As above mentioned, the mixture of sulphate of copper with dough makes a light bread out of an inferior grade of flour. This is a reprehensible act, since it introduces an irritant drug along with natural food, and may give rise to serious disturbances of the digestive functions. Kuhlmann has shown that a mixture of 1 part of sulphate of copper with 70,000 parts of damaged flour will improve its bread-making qualities. This amount of the copper salt cannot cause any material disturbance of health to those eating the bread; but, unfortunately, it has been shown that bakers use a much larger proportion than is necessary, and in consequence have done harm to their customers. One baker, in Germany, was shown to have used a mixture containing $\frac{1}{5000}$ part of sulphate of copper by weight, and in the bread of another baker the blue vitriol crystals were easily distinguished in the bread sold to the public.

It is seldom that these poisons are designedly administered with homicidal intentions, since their detection, both by the color and taste, is too easy to permit it. A husband attempted to poison his wife by adding verdigris to a dish of beans. The bad taste prevented her from eating them. He buried the cooked mess in his garden, from which it was disinterred, and then examined by chemists. They proved the certain presence of the metal. He was condemned to hard labor for life.[47] Cases of poisoning from these salts may, then, be divided into those in which a large dose is swallowed, either by accident or with a view to suicide, and those which proceed from the con-

[47] Jour. de Chimie, Chevalier, 1854.

tamination of food by unclean copper vessels, or by the salts of copper used as coloring matters for confectionery, etc., the injurious qualities of which will depend upon the amount of contamination.[48] If metallic copper is subjected to the action of water, acetic acid or vinegar, or hydrochloric acid, it is not affected by it unless the fluid is in contact with air or oxygen, so that copper vessels can be used for cooking, if the fluid be removed from the copper vessel while it is hot, in which case the vinegar, fatty acids, or water will have no effect upon the copper; but if the fluids be allowed to stand in the copper vessel for several hours when cold, air is absorbed, the copper is in part oxidized, dissolved by the acid, and impregnates the food. If metallic copper, in a very finely divided state, comes in contact with the mucous membranes, it becomes oxidized and absorbed, and gives rise to certain disagreeable symptoms. This is the so-called professional poisoning, which rarely occurs in workers in bronze, where very finely divided copper in the form of dust is inhaled.

In a very instructive article by Dr. Hönerkopf,[49] it is maintained that sulphate of copper cannot, in a strict sense, be considered a poison. He refers to numerous cases in which this medicine was taken in large or repeated doses without harm, and usually with benefit, and shows that the greater number of symptoms ascribed to it are not really observed.

Salts of copper, even in moderate doses, produce, after they are received into the stomach, and sometimes immediately after their ingestion, disturbances of digestion, such as pain at the pit of the stomach, nausea, vomiting, and abdominal, colicky pains, which are followed by diarrhea. These effects occur principally after the use of the sulphate, carbonate, and acetate of copper.

It should be remarked, also, that the irritating effect upon the mucous membrane does not hinder the absorption of the salts of copper, for these are found in the urine and the secretions from the lungs.[50] According to Miahle,[51] the salts of copper with the organic acids are more readily absorbed than those formed with the stronger or inorganic acids, since the precipitate produced by a combination of the latter with the proteid substances in the intestines dissolves quite slowly. It should be observed that, as in the case of the lead salts,

[48] Metallic copper is not poisonous, according to the best authorities, unless an acid has formed it into an irritating salt of copper.

[49] Casper's Vierteljahrsschrift, VIII. 212.

[50] Dangers and Flandin.

[51] Chimie appliquée à la Physiologie et a la Thérapeutique, Paris, 1856.

compounds of copper are not so easily absorbed into the system when taken in large as in small doses, since the large doses cause more local irritation, and hence are more rapidly eliminated or expelled by the natural outlets of discharge; the small doses, *per contra,* being longer retained in contact with the internal surfaces, are slowly, but more surely, absorbed into the circulation. Thus, as medicines, these salts are prescribed in small, unirritating doses, when the practitioner wishes to effect their absorption, and in large doses, where he wishes to evacuate offending materials from the primæ viæ; ¼ or ½ grain (0.16 to 0.32 gm.) is used in the former, and 2 to 10 grains (0.13 to 0.61 gm.) in the latter case, or for an emetic action.

Pecholier and Saint-Pierre,[52] in their observations of persons exposed to the absorption of verdigris, and, in corroboration of these observations, by experiments on animals, believe that its slow absorption favors the development of fat, and they noticed that the women in these factories were free from anemia and chlorosis. This opinion is at variance with the older writers on toxicology, who have taught that the slow absorption of the copper salts causes colicky pains similar to those in lead poisoning, as well as emaciation and constitutional disturbances. It may be said, however, that many cases of so-called poisoning by copper salts have also been simultaneously subjected to the absorption of metallic lead, or of arsenic in the form of Scheele's green (copper arsenite) Schweinfurt's green, Paris green, vert de Vienne, etc., which contain aceto-arsenite of copper. It is highly probable, therefore, that the ill effect of this exposure may be due to another agent than copper, and one upon which there is an agreement as to a definite poisonous action.

The use of copper utensils in the preparation of food has occasionally given rise to serious consequences, on account of the impregnation of the food by some salt of this metal. If the vessels are bright and clean, very little harm can possibly result from this cause, when ordinary articles of food are boiled in them, and not allowed to remain in them after they become cool. Saline, acid, or oily matters act, however, upon copper vessels, and if these are not clean, having been already exposed to moist air and become covered with the carbonate, the food may be impregnated with this poisonous salt in sufficient quantity to produce alarming symptoms. Such will be especially the case, if articles of the kind have been allowed to remain in

[52] Etude sur l'Hygiène de ouvriers employés à la Fabrication de verdet, Montpellier Médical, t. VII. 1864, p. 97.

the vessels to cool. It need hardly be stated that tinning the vessels is the only certain mode of preventing such effects, though here we have a further risk from the fact that copper is frequently not properly tinned, but is coated with an alloy containing metallic lead. In the latter case, the colic and poisoning due to lead may mislead the mind of the medical witness into the supposition that the symptoms were due to copper, when the true explanation would be that of lead poisoning.

There can be little doubt, we think, that the frequency of accidents from this cause is much exaggerated, and that in many cases the sudden illness, which is mistaken for the symptoms of copper poisoning, is really due to the unwholesome nature of the food eaten, or to other causes. We are led to this belief, not only from a consideration of the extremely small quantity of copper that in most cases can be dissolved, but also, as before stated, from the reflection that unwholesome food is capable of giving rise to a set of symptoms very similar to those produced by copper, and, finally, from the fact that in several cases of suspected poisoning by copper, this metal could not be detected by chemical analysis in portions of the food used.

Prof. Langenbeck, of Göttingen, reported an instance of the poisoning of thirty-one persons, who had eaten a portion of beef sausage. This sausage meat had been fried in lard, which had stood for two days in a badly tinned copper vessel, and was said to have become green in consequence. The poisoning was, therefore, attributed to copper. Dr. Paasch, in order to estimate the amount which each person in this instance must have taken, makes the following calculation. He assumes as barely possible that 1 sc. (1.3 gm.) of metallic copper could have been dissolved by means of the fatty acids existing in the lard. This amount would correspond to 25 grains of oxid of copper, 57½ grains of anhydrous, or 63 grains of crystallized acetate of copper. Supposing that the whole of this had been taken up by the food and entirely consumed, each person would have swallowed 2 gr. (.13 gm.) of verdigris.[53] That so small an amount should be capable of producing alarming symptoms of poisoning is hardly possible, when we reflect how much larger doses of this, and other equally poisonous salts of copper, have been given without harm in medical practice. Gerbier is said to have given the subacetate in doses amounting to 20 and even 30 grains in the twenty-four hours, and Solier de la Romillais 10 to 12 grains a day.[54] The sulphate of copper is frequently

[53] Casper's Vierteljahrsschrift, Jan. 1852.

[54] Guersent, Dict. des Sci. Med. art. Cancer.

given in doses of 15 gr. (.1 gm.) at a time, for the purpose of procuring emesis, in narcotic poisoning. Richmond gave as much as 1 scruple of carbonate of copper daily to patients suffering with obstinate, neuralgic affections, and Key, for the same purpose, administered as much as ½ ounce daily, divided into three doses, continuing the treatment for a fortnight, with no other result than the cure of the disease.[55] Pereira says that he administered 6 gr. (.4 gm.) of the sulphate of copper thrice a day for several weeks, in an old dysentery, without any other obvious effect than a slight nausea, and amelioration of the disease for which it was given. If the symptoms arising from the use of unwholesome food, such as sausages, old cheese, and the like, be now compared with those which are ascribed to poisoning by copper, a very great similarity will be found between them.[56] Dr. Paasch relates instances in which the conviction was so strong that the symptoms of poisoning must have been due to a salt of copper, that a chemical investigation of the food was undertaken, which resulted, however, in the fact that not a trace of copper could be discovered.[57] Dr. Taylor, in his work on poisons, states that he was required to examine the following case: "In an extensive poor-law union, a number of the paupers had been seized with diarrhea and dysentery, and several of them died. There was no apparent cause for this sickness and mortality; and it was suspected that the soup, which was daily prepared in large copper boilers, might have become impregnated with the metal, and have given rise to the symptoms, although these were scarcely indicative of irritant poisoning. I ascertained that the copper vessels were cleaned out daily, that the soup was made with salt and other vegetables, but was poured into other vessels to become cool." The soup, moreover, gave no trace of copper by the iron test, was unaffected by a current of sulphureted hydrogen gas, and the incinerated residue, after evaporation and calcining, gave no sign of the existence of copper with any of the tests.

While these considerations throw doubts upon the frequency of poisoning by food impregnated with copper from the use of cooking utensils of this material, they do not, of course, destroy the well-attested fact of its occasional occurrence. It appears evident, however, that the slightest attention to cleanliness in the keeping of such articles is all that is necessary to secure immunity from danger.

The use of verdigris or other salts of copper for the coloring of confectionery or other edible articles is manifestly a very pernicious

[55] Dict. de Méd. art. Cuivre.
[56] *Vide* Poisonous Food, Chap. VI.
[57] Loc. cit.

practice. A highly interesting case, in which a whole family was poisoned, and two of its members died, from the use of vegetables thus colored, is reported by Kramer.[58] Dr. Percival found a strong impregnation of copper in pickled samphire, of which a young lady ate, one morning, a considerable quantity, and which proved fatal in nine days. Dr. Falconer once detected so large a quantity in some pickled cucumbers, bought at a great London grocer's, that it was deposited on a plate of iron, and imparted its peculiar taste and smell to the pickles. It seems, indeed, to have been, at one time, the custom to make a point of adulterating pickles with copper; for, in many old cookery books, the cook is told to make her pickles in a copper pan. or to put some half-pence among the pickles to give them a fine green color.[59] Many of the cases of poisoning by confectionery are due to the arsenite of copper, or Scheele's green, which we have treated of elsewhere,[60] and are more likely due to arsenical poisoning.

Numerous cases are related, in which copper coins have been swallowed, with no symptoms of copper poisoning resulting. On the other hand, a case is mentioned by Dr. Jackson, of Boston, in which a half-cent, swallowed by a child, produced nausea and vomiting; another, by Dr. Budd and others;[61] and another by Dr. Hartshorne, in which a boy five years old died with all the symptoms of poisoning by copper, just two years after having swallowed a brass button.[62] A curious case is related by Deutsch, of a boy six years old, who swallowed a number of small copper coins. His medical attendant prescribed vinegar and other organic acids! In consequence of this singular treatment, he was seized with alarming symptoms, violent colic, and vomiting and purging of greenish-colored mucus. Finally, he was enabled to throw up the coins by means of an emetic of ipecacuanha, but recovered very slowly from the effects of the poison.[63] A curious question might well arise in such a case as this. Metallic copper is usually acknowledged not to be poisonous; the poisonous salt, *viz.*, verdigris, was here formed in the stomach by the administration of vinegar. Query: Was the poison administered?

There have been occasional periods of excitement among toxicologists and physiological chemists as to whether copper is a natural constituent of the body. Frey[64] states that "copper has been noticed in the blood, bile, and biliary calculi of man. It is excreted by the

[58] Canstatt's Jahresbericht für 1851, Bd. IV. s. 269.
[59] Christison, p. 352.
[60] *Vide* Arsenite of Copper, § 183.
[61] *Vide* Beck.
[62] Taylor, Am. ed. p. 112.
[63] Canstatt, 1851, Bd. IV. s. 269.
[64] Histology and Histochemistry of Man, Heinrich Frey, translated by A. E. J. Barker, N. Y. 1875, p. 62.

liver." Tardieu also discusses the question. MM. Bergeron and L'Hôte[65] record an examination to show that in all probability no copper is present in the tissues of the body unless accidentally introduced. They were induced to conduct their experiments, on account of the poisoning of two women at St. Denis, by Moreau, by means of copper, the presence of which was not discovered until revealed by chemical analysis, corrosive sublimate having been the suspected poison. It is probably a fallacy to suppose that copper is a normal constituent of the body, and with this explanation it may be stated that Orfila has detected traces of copper in the bodies of animals not poisoned by any of its preparations, and Wackenroder has obtained it from human blood. It has also been found in coffee, wheat, and flour, by M. Sarzeau and others.[66]

According to the calculations of Sarzeau, a man would introduce into his system by ordinary aliments 0.44 gm. (6.9 gr.) of metallic copper in the course of fifty years, not allowing that any of the metal is eliminated by the emunctories. "Though the results announced by chemists, and the narration even of their experiments, were of such a character as not to be accepted without discussion, they were still allowed entrance upon the rolls of science, and for several years it was found easier to accept, rather than to verify, them. This tendency soon became general. Along with copper were ranged normal arsenic, normal lead, normal manganese, etc. The reactionary movement, however, was not long in asserting, through the results of several chemists who found by careful experiments, putting out of their way possible source of error, that the animal system did not naturally contain these poisonous products. M. Roussin records an experiment upon the body of a soldier, killed suddenly by a fall from the barracks in Algeria, where he endeavored to determine the presence of normal copper in the organs and other tissue, with negative results.[67]

MM. Bergeron and L'Hôte[68] examined the liver and kidneys of fourteen bodies, and found traces of copper in all of them; in these analyses every precaution was taken to prevent the accidental introduction of copper during the analyses. These authors conclude that copper is always present in the liver and kidneys, and that the amount never exceeds 2½ or 3 milligrams in the liver, unless copper com-

[65] From N. Y. Med. Rec. April 17, 1875. p. 272.

[66] Vide Bley's Archiv. für Pharmacie. Oct. 1853; also Christison on Poisons, Am. ed. p. 356.

[67] Tardieu, op. cit.

[68] Jour. de Chimie Médicale, 1875, p. 115.

pounds have been taken as medicine, and rarely reaches the amount of 2 milligrams. MM. Bourneville and Yvón found in the liver of an epileptic,[69] who had taken during life 43 grams of ammonio-sulphate of copper as a medicine, 0.295 gram of copper, which corresponds to 1.166 grams of sulphate of copper, although the patient had taken none of the copper medicine for three months before death. These gentlemen made the analyses of the organs of the two females in the "Moreau" poisoning case, and found in the liver an amount of copper corresponding to 0.119 gram of sulphate of copper in one case, and to 0.084 gram in the other. They stated, therefore, that, since so large an amount of copper was found, and because the symptoms were not inconsistent with acute copper poisoning, and a careful post-mortem examination failed to reveal a natural cause for death, a compound of copper must have been the cause of death. L. M. V. Galippe[70] takes exception to the above conclusions of Bergeron and L'Hôte, and considers that an expert has no right to state that death has been produced by copper poisoning, when such small amounts as the above are found, since much larger quantities have been detected in cases in which death has been due to natural causes. Yvon found in the liver of another person, who had been treated during life with copper compounds, 0.236 gram of metallic copper; Rabuteau found 0.239 gram; and Galippe found 0.310 gram, 0.220 gram, and 0.120 gram of metallic copper in different cases. From experiments upon animals and men, Galippe concludes that (1) except in cases of suicide, acute poisoning by compounds of copper ought not to be possible, both on account of the horrible taste which these compounds have, and on account of their emetic property, which causes the expulsion of the poison; and (2) poisoning of a mild grade is impossible, since, according to his experiments upon animals, when small doses are given, tolerance of the system for the copper compound is established without any injurious influence upon the health. (see § 295.)

290. Effects on workmen in factories where copper is used.—Desayure[71] relates that the workmen, exposed to the manufacture of copper, after a certain time show symptoms of nausea, vomiting, stomachal disturbance, diarrhea, oppression, and fever, as well as intestinal distress, meteorism, and habitual constipation. Perron[72] adds, in addition to the above effects, that these successive morbid

[69] *Ibid.* p. 236.

[70] Etude toxicologique sur le cuivre et ses composés, Paris, 1875.

[71] Etude sur la maladie des ouvriers de la manufacture d'armes de chatellerault. Ann. d'Hygiène et de Médecine Légale, 1861.

[72] Des maladies des ouvriers produites par le cuivre et l'absorption des molécules cuivreuses. *Ibid.* 1861.

conditions predispose to the development of pulmonary consumption, —especially in those whose temperament and constitutional tendencies are towards this disease. As a rule, however, in cases of copper poisoning, no lesion of the lungs can be detected to account for the cough and night sweats from which these patients suffer. In our opinion these symptoms and dangers are more likely to be caused by the presence of fine, metallic dust in the respired air, than to any poison caused by the absorption of metallic copper into the circulation. A similar example is offered of the fine particles of diamond powder, corundum, iron, and other similar substances, which irritate the pulmonary and the gastric mucous membrane. A consultation and study of conflicting authorities led Maisonneuve[73] to express the following conclusions in reference to the so-called "copper colics":—

"1. Work and manipulation of cold, metallic copper are innocuous; but in workshops, where the metallic particles of oxid or salts of copper abound in the air, the inhabitants may receive this irritating dust into the air passages, and cause oppression and very intense dyspnea (asthma?) with bronchial and laryngeal spasms.

"2. Copper colic exists, though it is denied by some authorities; it is, however, of short duration and of very slight consequence."

Maisonneuve describes this colic as characterized by a painful sensation, having its location at the pit of the stomach or at the navel, and extending over the region of the transverse colon, being increased by pressure; it causes nausea and vomiting, and rarely diarrhea. These symptoms have a very short duration, and the workmen never seek for medical relief, but treat themselves by drinking milk in large amount. From this last-named, successful means of treatment, one is led to the conviction that the so-called colics are caused by local irritation from the presence of a mechanical irritant, because the effects of this irritation are so readily allayed by the reception of an unirritating substance, such as milk would seem to be.

It will be seen by comparison with the description of lead colic, that the colic caused by copper is very different. A purple line also appears at the junction of the teeth and gums, at the same place as the blue line of lead poisoning, and due to the same cause, *viz.*, the formation of the sulphid of the metal.

As has been previously mentioned, when death is caused by the poisonous, or rather irritating, action of sulphate or other salt of copper, it follows the ingestion of a large amount of the agent, and in this

[73] Hygiène et pathologie professionelles des ouvriers des arsenaux maritimes, ouvriers en cuivre. Arch. de Méd. Navale, 1865, p. 25.

case the evidence of irritation or inflammation, as well as the amount of copper in the primæ viæ, must be large in extent or amount. The fact must not be overlooked that sulphate of copper is often used as an emetic in cases of suspected poisoning.

291. Symptoms of acute poisoning.— The symptoms of acute poisoning from copper salts come on very soon after the ingestion of the poison. There are violent headache, vomiting and purging, severe colicky pains, eructations, salivation, cramps in the limbs, and finally, convulsions and insensibility. The vomitus, after large doses, has a green color, and is colored azure-blue by ammonia water. The diarrhea is usually like that of dysentery. Sometimes jaundice is observed. In a case related by Dr. Percival, 2 dr. (7.8 gm.) of sulphate of copper produced fatal convulsions. In another, where the same salt was swallowed, there were no convulsions. The child, which was sixteen months old, died in four hours.[74] Those cases which have terminated fatally have lasted a variable period. Thus, in one reported by Pyl, a woman, who swallowed 2 ounces of verdigris, died in three days; in another, by Neumann, ½ ounce destroyed life in sixty hours; and in another, in which 1 ounce of blue vitriol was taken, death ensued within twelve hours.[75] In most cases, however, of poisoning with these salts, the patient has recovered, when timely and efficient means have been used.

In those cases in which the poison has been conveyed accidentally, through articles of food, into the system, the symptoms have been the same as those mentioned, although they have usually not come on until a few hours afterwards. They are thus described by Orfila: "An acrid, styptic, coppery taste in the mouth; parched and dry tongue; a sense of strangulation in the throat; coppery eructations; continual spitting; nausea; copious vomiting, or vain efforts to vomit; shooting pains in the stomach, which are often very severe; horrible gripes; very frequent alvine evacuations, sometimes bloody and blackish, with tenesmus and debility; the abdomen inflated and painful; the pulse small, irregular, tense, and frequent; syncope, heat of skin, ardent thirst, difficulty in breathing, anxiety in region of the præcordia, cold sweats, scanty urine, violent headache, vertigo, faintness, weakness of the limbs, cramps of the legs, and convulsions." Such are the symptoms which, it is said, are produced by the ingestion of articles of food contaminated with copper salts. How far they are really due to this cause we shall presently inquire.

Kobert presents in brief the following symptoms of acute poison-

[74] Med. Gazette, Vol. XVIII. p. 742. [75] Quoted by Beck.

ing by copper salts: "Green scabs in mouth, green vomit, disagreeable metallic taste, salivation, sensitive and inflated abdomen, brownish-red diarrhea, small pulse, cold skin, icterus (jaundice), paresis of limbs, and collapse." The symptoms of poisoning by a copper salt usually appear ten minutes after its ingestion.

292. **Lethal dose.**— Three ounces (about 93 gm.) of the acetate of copper, and a little more of the sulphate, may be considered a fatal dose.

293. **Treatment.**— Administration of a solution of ferrocyanid of potash, followed by washing of the stomach, as this will produce an insoluble copper ferrocyanid; albuminous drinks, milk or white of egg mixed with water, oxid of magnesia; later on, hot or Turkish baths and electricity, the latter for stimulating the muscular contractions.

294. **Action.**— On animals. Bernatzic[76] likens the poisonous properties of copper salts to those of zinc and silver; silver, copper, and zinc are allied much in their action to each other, yet copper, a little more poisonous than the other two, if taken in sufficiently large quantities, will interfere with the functional activities of the muscular and nervous systems. In consequence of this interference, development of cell growth is prevented, and the number of the red corpuscles is diminished, which will prevent the nutrition of the tissues (metabolism), resulting in cachexia. These salts do not, therefore, cause serious changes of the tissues, or permanent disturbances, nor do fatal effects follow their ingestion, as after other poisonous metals, such as lead, mercury, arsenic, antimony, and phosphorus; for, on intermission of the administration of the copper poisons, the injurious effects cease, and the functions again become normal.

The effects on man are very similar to those on animals. The green vomited matters may be distinguished from bile by the addition of ammonia, which changes the green into a blue color.

295. **Chronic poisoning.**— The chronic poisoning by salts of copper is a matter of dispute. Experiments would throw doubt upon the production of chronic poisoning from the repeated, small doses taken for a long time; apparently, the authorities, supporting the theory of chronic poisoning by copper, have confused the evidence of poisoning with the symptoms which are usually classed as following the administration of other metals which are adulterations of the copper, such, for instance, as arsenic and lead. Sheep have been fed with vines

[76] Encyc. d. ges. Heilkunde, XI. s. 433.

which have been treated with copper sulphate; oxen and pigs have been fed on grass treated with a 3 per cent solution of copper sulphate, without in the least suffering any disturbance of their health. Mach[77] has fed cows with green fodder containing as much as 200 milligrams of copper sulphate, for long periods of time, and without discovering the slightest injurious action. It is the opinion of the latest and best authorities that there is no chronic poisoning among men or animals from the administration of continuous, small doses of copper salts mixed with their food; the liver, kidneys, and other organs excreting the copper by means of the bile and the urine, and thus preventing the accumulation of a sufficient amount of the poison to interfere with normal condition. This theory is still further supported from the fact that the kidneys and liver do not appear to lose their functions when these organs are normal. If, however, these organs are in an abnormal state, and the excretion of copper is thus prevented, injuries to the general health may follow. Even Kobert, who gives the symptoms of chronic poisoning as a pale green appearance on the gums, copper edge on the teeth, copper colic, catarrhal inflammation of the stomach and intestines, marasmus, and a questionable copper paralysis, apparently, would incline to the belief that these last derangements of the nervous system may be due to impurities such as arsenic and lead.

296. Diagnosis.—Kobert gives the following as a diagnostic sign of poisoning by copper salts: The green discoloration of hair, a peculiar muddy complexion of the skin, and cachexia, the last being also a diagnostic symptom of lead poisoning.

297. Post-mortem appearances.—The mucous membrane of the stomach and intestines is inflamed and thickened, in some places eroded, and, in a case quoted by Orfila, the small intestine was perforated. If the patient has not survived long, the mucus of the intestines will be found tinged of a green color. The digestive tube is generally distended by an enormous quantity of gas, and the mucous membrane of the alimentary canal is often red and inflamed, sometimes throughout its whole extent; at other times some ecchymoses distributed in the submucous cellular tissue are noticed, and also ulcerations and gangrenous patches. But the most important and remarkable appearance is the predominance of inflammatory lesions over blood extravasations, in fact a true catarrhal inflammation such as might be caused by any mechanical irritant. These latter rarely

[77] Bericht ueber die Ergebnisse der im Jahre 1886, ausgeführten Versuche zur Bekämpfung der Peronospora, St. Michele, Tyrol.

occur, and are met with in exceptional cases, under the serous coat of the intestines, lungs, and heart. (Tardieu.)

No other changes worthy of note have been observed. A fatal case is reported by Mr. Cockburn,[78] in a woman who swallowed 2 or 3 drams of sulphate of iron with 7 of sulphate of copper. The symptoms were such as described above, and death took place in about twenty-four hours. Yet there was no disorganization whatever of the mucous coat of the stomach and intestines. We must admit that fatal poisoning from copper is so rare an occurrence,—especially in its chronic form,—that, in the older cases of record, there is considerable doubt whether the copper ingested by the victim was not adulterated by imperfect smelting with arsenic and lead; consequently, the post-mortem appearances, which are attributed by writers on toxicology, confuse the effects produced by copper with those of arsenic and lead. In the early edition of this work on poisons we stated that Dr. Taylor[79] described chronic poisoning by copper, in which, sometimes, the patients die from fever and exhaustion.[80] Again, many of the cases of copper poisoning, reported in earlier works on toxicology, contain records of cases of poisoning, from Scheele's green, Schweinfurt's green, as well as cupric sulphate and verdigris, the first two being compounds, respectively, of arsenic. Consequently, some of the symptoms and post-mortem appearances described by writers closely simulate the effects of copper, with those of lead and arsenic and chromate salts.

298. Chemical examination.—The tests for copper are quite delicate, and there is usually no difficulty in detecting it, when present in very small amounts. The principal tests for copper compounds are the following: 1. The color of copper salts is either green or blue, and metallic copper has a peculiar odor, which can be perceived by rubbing the moistened finger upon a copper coin. 2. If nitric acid be added to metallic copper, it readily dissolves it, and reddish vapors are evolved, which can be easily perceived by looking down into the vessel in which the test is performed. 3. A solution of potassium hydrate produces a bluish-green precipitate of the hydrate of copper, which is soluble in ammonia water, and in some organic fluids, such as glycerin and tartaric acid. 4. Ammonia water, if added to a solution of a salt of copper, gives, at first, a precipitate of the hydrate

[78] Lancet, Aug. 1856, p. 248.

[79] On Poisons, p. 520.

[80] *Vide* also, Böcker, Vergiftungen, 1857, p. 42; Orfila, Toxicologie, I. 912; Casper's Vierteljahrsschrift, 1852; I p. 79; II. p. 22: 1856, II. p. 41; 1857, II. p. 228; Ann. d'Hygiène, 1847, I. 392; and Avril, 1858, p. 328; Dublin Hospital Gazette for Sept. 1854; Lancet, Jan. 1855.

of copper; but if an excess of the ammonia is added, the hydrate is redissolved, and an azure-blue solution results. 5. A current of sulphureted hydrogen passed through a solution of a salt of copper, produces a brownish-black precipitate of the sulphid of copper, which is readily soluble in nitric acid. 6. A solution of ferrocyanid of potassium added to a solution of a salt of copper, produces a reddish-brown precipitate of the ferrocyanid of copper, which is insoluble in hydrochloric acid. 7. A piece of metallic iron introduced into a solution of a salt of copper which has been acidulated with hydrochloric acid, becomes coated with a deposit of metallic copper, which can readily be recognized by its color and physical properties. A steel needle or a knife blade is usually the most convenient form of polished iron to use, and this deposit of copper upon the steel should always, in legal cases, be taken to court as one of the *corpora delicti,* since the jury can easily recognize metallic copper. 8. The electrolytic test, like the foregoing, has for its object the separation of metallic copper from its solutions. It is performed by placing the solution in a platinum vessel, connecting this with the cathode of a galvanic battery, acidulating the solution with hydrochloric acid, and immersing in this solution a bit of platinum wire, to serve as the anode. This wire must be carefully protected from touching the platinum dish. In twelve to twenty-four hours all the copper will be deposited upon the inside of the dish. The dish is washed, dried as quickly as possible with alcohol and ether, and weighed. The gain in weight, of course, is the weight of the copper in the solution tested. The copper can then be recognized by its physical properties, color, etc., and by dissolving it in nitric acid.

299. Separation from organic mixtures.— The organic matter must first be destroyed, either by the method described for the isolation of arsenic, or by burning up the organic matter in a crucible, after the addition of pure saltpeter. After precipitation of the sulphid of copper by sulphureted hydrogen, the precipitate should be dissolved in nitric acid, the resulting solution evaporated to dryness upon a water bath, the residue of nitrate of copper dissolved in water, after the addition of a drop or two of hydrochloric acid if necessary, and this solution tested by the special tests given above.

XXII. Chromates.

300. In general.— Chromic anhydrid and three of the salts of its hypothetical acid, H_2CrO_4, the normal chromate and bichromate of

potassium, and the chromate of lead, are the chief sources of chromate poisoning.

Chromic anhydrid, CrO_3, often improperly called chromic acid, is the anhydrid of a hypothetical acid, H_2CrO_4, which forms salts of the type formula X_2CrO_4. It occurs in large saffron-colored or crimson prismatic needles, is deliquescent, freely soluble in water with an acid reaction, and may then be considered as a solution of chromic acid. It is produced by the action of sulphuric acid upon potassium bichromate.

Potassium chromate, K_2CrO_4, forms large, yellow, rhombic crystals, soluble in water. It is used in the manufacture of lead chromate, which is extensively used under the name of "chrome yellow" as a yellow pigment in dyeing.

Potassium bichromate, $K_2Cr_2O_7$, is an orange-red, crystalline body, soluble in water, with a bitter, metallic taste, insoluble in alcohol. It is used quite commonly in dyeing, in stains for furniture, and as an ingredient of several "battery fluids." It is also used in the manufacture of other chromates, and in medicine as an escharotic.

Lead chromate, $PbCrO_4$ known as "chrome yellow," is largely used as a pigment in dyeing, and also as an artificial, coloring agent for confectionery and various food stuffs. It is insoluble in water and dilute acids, but soluble in strong alkalies.

The neutral chromate of potash, the bichromate of potash, and the chromate of lead are the only salts of chromium of importance in toxicology. The chromic acid has produced one death, as reported by Mr. Blyth in his table of ten years of mortality, and four accidental deaths from bichromate of potash. Falck has found, in medical literature, ten accidental deaths from the bichromate salt, six suicidal, and one in which it was used as an abortifacient. In the process of manufacturing bichromate of potash, workmen have suffered from what Sir B. W. Richardson describes under the name of "Bichromate Disease." This is caused by the inspiration of particles through the nose or mouth, which produces a bitter and disagreeable taste with an increase of salivation, the saliva thus eliminating the poison; if the mouth is closed and the inspiration is through the nose, the nasal septum becomes inflamed, gets thin, and finally ulcerates, and may be wholly destroyed. The bichromate also causes painful skin affections and persistent ulcerations, which the workmen call "chrome-holes;" if the skin is unbroken, the local effect is slight, if at all. When, in dye factories, the salt gets into a wound or crack in the leg of a horse, ulceration is produced, and these horses may lose their hoofs.

301. Chromic acid.— Chromic acid (chromic anhydrid), whose only use in medicine is as an external caustic, has been swallowed accidentally, in the case reported by Tisne.[81] In this case a dentist applied it to a fungous growth in the mouth, which was followed by a burning taste in the throat, pain in the neck, a persistent vertigo with the vomiting of a ropy-green fluid, and, finally, by great prostration. The patient recovered, though chromium was found in his urine. In another case reported by White,[82] the application of a solution of 50 grains in ½ ounce of water, to the external genitals, for the removal of papillary vegetations, was followed by poisonous symptoms, from which the woman died in twenty-seven hours. The kidneys and the liver were found congested, and chemical tests showed the presence of chromium in their tissues. Blyth refers to a case of poisoning by the chromate of potash (Maschka), in which a woman, aged twenty-five, committed suicide by swallowing a piece of chromate of potash (estimated to weigh 6 gm.), with symptoms of vomiting, diarrhea, pain in the stomach, rapid collapse, and, finally, death fourteen hours later. In another case recorded by MacNiven,[83] a man swallowed a lump of potassium bichromate estimated to weigh 2 drams (7.7 gm.). He recovered after the following symptoms which began in fifteen minutes: Lightness in the head; sensation of great heat in the body, followed by cold sweat; vomiting in twenty minutes, pain in the stomach, giddiness, specks before the eyes, insatiable thirst, and loss of power over the legs. On admission to the hospital two hours after swallowing the poison, his pupils were dilated, the face was pale and cold, and the pulse feeble; he suffered from intense epigastric pains and a feeling of depression, and there was some stupor. The stomach was emptied by emetics and the stomach pump, the patient was given tepid emollient drinks, subcutaneous doses of sulphuric ether being administered.

302. Bichromate of potassium.— This salt, being extensively used in dyeing and electric batteries, has given rise, in several instances, to accidental poisoning. Locally applied, its action is irritant, causing, in the workmen who use it, troublesome sores and ulcerations upon the hands. Taken in poisonous doses internally, its action is highly irritant also, and death has been caused by it, with the symptoms usually attending the action of irritant poisons. Mr. Wilson, however, relates a case in which death was produced by it, without any vomiting or purging having occurred.[84] Several fatal cases have occurred

[81] Jour. de Méd. Paris, 1887.
[82] London Lancet, Sept. 22, 1873.
[83] University Med. Mag. 1889.
[84] Med. Gazette, Vol. XXXIII. p. 734.

in Baltimore. The following was communicated by Dr. Baer to Professor Ducatel: A laborer, aged thirty-five, received a small quantity of the solution into his mouth on attempting to draw it off from a refiner by exhausting the siphon by suction. His first impression was that he had spit it out; but only a few minutes elapsed before he was seized with great heat in the throat and stomach, and violent vomiting of blood and mucus. The vomiting continued until just before his death, which occurred in five hours. On section, the mucous tissue of the stomach, duodenum, and about a fifth of the jejunum, was found destroyed in patches. The remaining parts of it could be easily removed by the handle of the scalpel.[85] A boy, who swallowed about 2 ounces of bichromate of potassium, was seized in half an hour with vomiting, and became almost totally insensible. He was pale and collapsed, the pupils were dilated and fixed, the pulse feeble, and he had, also, cramps in the legs. An emetic of sulphate of zinc was given, and the stomach pump used, until the pinkish color of the washings obtained by it had ceased. He had an attack of gastrointestinal inflammation, from which he did not recover for four months.[86] Dr. A. McCrorie[87] reports the following case of poisoning by bichromate of potassium. A child twenty months old swallowed a piece of "red chrome" about 5 or 10 grains in weight, and was found semi-comatose with a livid complexion. The eyes were half closed and pupils were dilated; the respiration was difficult and wheezy, the pulse almost imperceptible. There was tenderness on pressure over the abdomen, and very marked at the pit of the stomach, causing him to cry and draw up his legs. He also had vomiting and purging. He improved somewhat, but two hours later he again became comatose, fell into a state of collapse with coldness of the surface, and was bathed in a cold and clammy sweat. He died in nine and a half hours after swallowing the poison. A man[88] sixty years old swallowed 3 drams (11.6 gm.) of bichromate of potassium dissolved in cold water. In quarter of an hour afterwards, he showed the signs of irritant poisoning, such as vomiting and purging, accompanied by violent, abdominal pain. When first seen by Dr. McLachlan, two hours after swallowing the poison, he was reclining in a chair, moaning, and complaining of these cramps; surface of body cold, with shriveled hands and feet, the skin wrinkled and dusky, like the collapse observed in cholera. The pulse was weak and barely perceptible, respiration was

[85] Beck, Vol. II. p. 666.
[86] Guy's Hosp. Rep. 1850, p. 214.
[87] Glasgow Med. Jour. May, 1881.
[88] *Ibid.* July, 1881, reported by Wm. A. McLachlan.

hurried, and urine was suppressed. His mental faculties were unimpaired. After a stimulating treatment, and application of heat, he gradually improved, and two days later was well, except that his urine and skin were yellow. He fully recovered. The suppression of the urine is probably due to inflammation of the kidneys produced by the chromic acid. Experiments upon animals have shown[89] that, after the subcutaneous injection of chromic acid, animals suffer from vomiting, diarrhea, albuminuria, and finally die in a few days; after death, inflammation of the kidneys is found. The same results were produced in rabbits by the injection of a neutral chromate (yellow chromate of potassium).

303. Chromate of lead.— (Chrome yellow.) Although this substance is insoluble in water, and, under many circumstances, in the stomach and intestinal fluids, sometimes it gives rise to acute poisoning owing to its decomposition after it enters the body. That such a decomposition of the chrome yellow does take place is shown by the fact that in one case of poisoning, reported by R. C. Smith,[90] chromic acid was detected in the urine; this was a case of professional poisoning, the patient being employed in weaving yarn colored with chrome yellow.

The frequency with which this poison is used as a pigment for coloring articles in common use, such as children's playthings, and even confectionery, shows the importance of a consideration of this substance, and is the apology for the introduction of the following extracts of several cases of poisoning by it made by the writer for the Boston Medical and Surgical Journal.[91]

"Two fatal cases of acute poisoning by chrome yellow are reported by Dr. von Linstow.[92] These cases occurred in children aged, respectively, one and three-quarters and three and one-half years, and the poisoning was caused by sucking an unknown number of small, yellow substances, which had been used for ornamenting pastry, and which consisted of gum tragacanth and chrome yellow. A piece, measuring 13x5 millimeters, yielded, on analysis, 0.278 gram of gum tragacanth, and 0.0042 gram of chromate of lead.

"Chromate of lead, on account of its insolubility, has never been considered an active poison, and the fact that it is used so largely as a pigment for coloring, not only ordinary substances, but also children's playthings, and even articles intended for food, such as con-

[89] Gergens, Arch. f. Exp. VI. p. 148.
[90] Brit. Med. Jour. 1882, p. 8.
[91] July 9, 1874, and Feb'y. 7, 1878.
[92] Vierteljahrssch. für gericht. Medicin, Jan. 1874.

fectionery, etc., renders these cases of more than ordinary interest. The extent of its use in confectionery can be seen by an examination of the report of analyses of confectionery, by H. B. Hill.[93] Thus, 77 samples, both white and colored, were analyzed; 21 were colored yellow, and in 17 of these the pigment consisted entirely of chrome yellow, in 2 partially; of 12 specimens, which were of an orange color, the pigment in 9 consisted entirely of the chromate of lead, and in 2 partially; 7 specimens of green were examined, 6 of which contained chrome yellow mixed with Prussian blue in five specimens, and with Scheele's green in the other. Of the 77 specimens examined, 36, therefore, contained the chromate of lead.

"The symptoms of poisoning did not commence until several hours after the ingestion of the chrome yellow, which took place between 9 and 11 A. M. Both children were taken sick at the same time (between 2 and 3 P. M., of the same day) with vomiting, which lasted for several hours. The vomitus was yellow in color. There were great prostration and extreme thirst, but no diarrhea and no pain. On the second day, both had a hot and red countenance, and were stupid. The younger, about twenty-four hours after the commencement of the symptoms, had a slight diarrhea and convulsions, which continued until death, which took place in forty-eight hours. On the third day, an erythematous eruption appeared on the chest and abdomen of the elder. He was dull and stupid, and the temperature in the axilla was 39.5° C. On the fourth day, the pulse and respiration became irregular, the breath extremely fetid, stupor and unconsciousness came on, and the patient died five days after the ingestion of the poison.

"After death, the mucous membrane of the stomach and duodenum was found swollen and loose, so that it could easily be raised from the submucous tissue; it was inflamed, as was also that of the œsophagus, throat, and larynx. In some places, the mucous membrane of the stomach and duodenum was entirely destroyed, and in one spot perforation had taken place, showing that the chrome yellow had a corrosive action. These appearances were probably not caused by the chromate of lead, as such, but by soluble compounds formed after the pigment had lain in the stomach some time, and had been decomposed.

"Besides the above appearances, there were found also hyperemia of the brain and its membranes, beginning fatty degeneration of the liver, commencing icterus, hyperemia of the kidneys, suppurative

[93] Mass. State Board of Health Report, 1873, p. 390.

pyelitis, and a softened spleen; conditions which are often seen after death from poisoning by other corrosive poisons.

"The number of these yellow ornaments ingested by the children could not have been more than six, since only seven were given them to play with, and one was afterwards recovered. If each child had eaten three of these, the fatal dose was less than 0.01 gram (between 1/5 and 1/6 of a gr.) of the chromate of lead."

"Leopold reports five cases of this form of poisoning, one of which proved fatal. The patients were employed in weaving cloth colored with chrome yellow (chromate of lead), which was quite loosely applied to the thread, so that a portion of the pigment was easily detached and became diffused throughout the air of the room. The patients were affected with a yellow-coated tongue, yellow sputa, loss of appetite, malaise, in some cases vomiting, pain in the region of the stomach and umbilicus, obstinate constipation and debility. The feces were yellow. These symptoms disappeared in a few weeks after the removal of the cause, except in the case of an infant nine weeks old, who died in six or eight days after the beginning of the symptoms, which, however, did not appear until about three weeks after exposure to the infected atmosphere. The symptoms in this case were fever, restlessness, shrieking, several yellow-fluid stools daily, redness of the skin over the chest and abdomen, parched lips, and, just before death, short respiration.

"After death there were found inflammation and perforation of the stomach, the same appearances which were seen in the two cases previously reported by Dr. von Linstow,[94] caused by ingesting the chrome yellow. None of the poison could be detected in any of the organs except the lungs, in which 3.6 milligrams were found.

"This is the third fatal case of chrome-yellow poisoning reported within a few years."

304. Symptoms of chronic poisoning.—In addition to those described in our introductory section may be noted the following, which occur to those workmen who handle the dry chromate salt used in dyes: Ulceration of the hands, arms, penis, septum of the nose. These symptoms are followed or associated with bronchitis, inflammation of the eyes (conjunctivitis), and inflammation of the kidneys (nephritis). Except when these ulcerations destroy the tissue, chronic poisoning is usually recovered from,—especially if the acute nephritis is treated, and the elimination of the poison accomplished by suitable treatment to assist the kidneys in eliminating the poison.

[94] See above, § 303.

305. Lethal dose.— 15 grains (1 gm.) of the bichromate of potash; much larger doses of the salts of the oxid.

306. Treatment.— Administration of the bicarbonate of sodium in solution, and the washing out of the stomach with the same; internal administration of carbonate of magnesium, or acetate of lead and diuretic medicinal treatment.

307. Action.— Effects on animals: Gmelin found that the chlorid of chromium was less active than the neutral chromate of potash, which, Mr. Blyth remarks, is not strange, because in this combination we get the action of two metals, chromium and potassium; 3 grams of the chlorid salt was followed by no poisonous action, whereas, 2 grams of the neutral chromate caused death within two hours. Gergens and Posner[95] found that the subcutaneous doses of .2 to .4 gram of the neutral chromate acted with great intensity on rabbits, which, after the injection, were restless and suffered from difficult respiration; death took place in a few hours.

That local, intestinal irritation causes diarrhea, when the poison is administered by the mouth, would seem to be shown by the fact that no diarrhea follows the subcutaneous injection in animals; the administration to animals by the mouth, in doses of about 2 grams, invariably produces diarrhea. This same dose, injected under the skin of a dog, was followed by inappetence, and a dry exanthematous eruption on the back in about six or seven days, with the hair dropping off in patches; in these cases there was no diarrhea or vomiting. The action of bichromate of potash appears to present powerful irritation to the stomach and intestinal canal; and, after its absorption, albuminuria, hemoglobinuria (bloody urine), and emaciation occur.

308. Diagnosis.— In acute poisoning, by the characteristic, yellow or red discoloration of the mouth; in chronic cases, ulceration of the nasal septem, skin, and frenum penis, albuminuria, and hemoglobinuria.

309. Post-mortem appearances.— The gross appearances are ulceration and discoloration of the mucous membranes of the mouth; ecchymoses and ulceration of the mucous lining of the stomach, as also of the intestines,—especially of the small intestine; fatty degeneration of liver and heart; exudation and necrosis of the epithelium of the convoluted tubules of kidneys. In chronic cases, rhinonecrosis, ulceration of skin, and bronchitis. Microscopical examination shows, in the epithelium of the convoluted tubules, a yellow cloudiness, the

[95] Arch. f. exper. Pathol. u. Pharm. Bd. 6, Hft. 1 and 2, paragraph 333, 1880.

outline of the cells being irregular and jagged. The glomeruli are moderately injected, the capsules containing an albuminous exudation; the canaliculi are filled with round cells imbedded in a liquid which coagulates on heating.

In the case of the woman above quoted, who poisoned herself with potassic chromate, the stomach contained more than a liter of dark chocolate fluid having an alkaline reaction; the mucous membrane near the cardiac and pyloric openings of the stomach was swollen and red in sharply defined patches; portions of the epithelial layer were detached; the rest of the mucous membrane had a yellow-brown color, and the intestine, throughout its whole length to the sigmoid flexure, contained a bloody, treacle-like fluid; the mucous membrane throughout its entire length was swollen, had numerous extravasations, and in portions was detached.

310. Tests and detection.—The tests for chromic acid are: 1. Chromic acid and chromates are either red or yellow, and, when soluble in water, give red or yellow solutions. 2. Sulphureted hydrogen changes the color of such solutions, if previously acidified with hydrochloric acid, from yellow or red to dark green, owing to the formation of the sesquioxid of chromium; which change may also be caused by other reducing agents. 3. A solution of chlorid of barium causes, in aqueous solutions of chromates, a yellow precipitate of chromate of barium, which is insoluble in acetic acid, but soluble in dilute hydrochloric or nitric acid. 4. A solution of sugar of lead produces a yellow precipitate of chromate of lead (chrome yellow), which is insoluble in nitric acid but soluble in potassic hydrate. 5. Nitrate of silver produces, in aqueous solutions of a chromate, an orange-red precipitate of chromate of silver, which is soluble both in nitric acid and ammonia water, and which is not produced in the presence of chlorids until all of the chlorin has first been precipitated as chlorid of silver. 6. Solutions of bismuth salts produce a yellow precipitate of chromate of bismuth, soluble in nitric acid, but insoluble in potassic hydrate. 7. A solution of a few drops of a chromic acid solution, and a little ether, to a very dilute, acidified hydrogen dioxid solution, yields a beautiful blue color.

Chrome yellow, when heated with carbonate of sodium upon charcoal with the blowpipe, yields a globule of metallic lead. It is insoluble in dilute nitric acid, but soluble in potassic hydrate, thereby differing from the yellow chromate of bismuth. When fused in a crucible with sodic carbonate and niter, chrome yellow is decomposed, chromate of the alkali and carbonate of lead being the result; these

can be separated by treating the fused mass with water, which will dissolve the chromate of the alkali, and will leave undissolved the carbonate of lead; the filtrate may then be tested for chromic acid, and the undissolved portion for lead, as given under § 284.

The same method may be used for the destruction of the organic matter as for lead and arsenic. By that process the chromic acid will be converted by the sulphureted hydrogen into the sesquioxid of chromium, which will remain in solution and must be precipitated from the filtrate, after boiling off the excess of sulphureted hydrogen, by ammonia water in the form of the hydrate of the sesquioxid of chromium. This can be collected upon a filter paper, dried, fused in a crucible with carbonate of sodium and niter, the fused mass extracted with water, and the tests for chromic acid performed with the filtrate, as mentioned above. The lead would be separated as described in speaking of the salts of lead.

In the case of small amounts of colored substances, such as colored spices, confectionery, etc., the material can be at once fused with carbonate of sodium and niter, which will destroy the organic matter and leave the chromic acid and the lead as mentioned above.

XXIII. Iron.

311. In general.— The compounds of iron which are of toxicological interest are chiefly the chlorid of iron and the sulphate.

Ferric chlorid, Fe_2Cl_6, is a yellowish-brown, amorphous body, slightly deliquescent, very soluble in water, with an acid reaction. It is used in medicine both in aqueous and alcoholic solution.

Ferrous sulphate, green vitriol $FeSO_4 + 7H_2O$, occurs in pale green, oblique, rhombic, prismatic crystals, very soluble in water, with an astringent taste, insoluble in alcohol. On exposure to the air it effloresces and oxidizes.

312. Preparations.— The many preparations of iron and its compounds, ordered by the United States Pharmacopœia, do not require very much comment in a work on toxicology. The most important of those preparations which do concern us in this treatise are: Ferrous sulphate; ferrous iodid; iron and quinin citrate; chlorid of iron; solution of sulphate of iron; syrup of ferrous iodid; citrate of iron and strychnin containing 1 per cent of strychnin.

313. Administration of iron in disease.— While the administration of iron in disease does not belong to a work on poisons, yet a few words in regard to treatment in serious anemia may not be out of

place, because, in these abnormal conditions, sometimes symptoms occur from which the patient may suffer, more than from the effects of his original condition, to relieve which, his physician may have prescribed iron preparations. A large portion of an iron preparation given as medicine passes out of the system without being absorbed. These cases, in which, associated with the anemia, there is an impairment of the process of digestion and a diminished intestinal nutrition, may be caused by the presence of syphilitic or cancerous cachexia. In these conditions the administration of iron does not generally repair the anemic condition, but it produces further disturbance of the digestion, accompanied with a general restlessness, and distress of the human economy. A furred tongue, gastric irritation, and a diminished action of the functions of the liver are particularly remarked. Moreover, in chlorosis and serious anemia, if iron is administered to relieve the bloodless condition of the tissues, very small doses of unirritating preparations of iron should first be given, and constipation corrected.

To show in what way iron may sometimes produce serious effects, we make no apology in presenting the results as derived from professional experience, and from a reliable therapeutical work.[96] "Small doses of the insoluble and less irritating preparations of iron produce, at first, no marked symptoms, except some improvement of appetite and a tendency to constipation. If the treatment is long continued, or larger quantities and the more irritating preparations are used, there appears, according to the susceptibility of the patient, decided gastric irritation, as shown by a furred tongue, a sensation of weight in the stomach after eating, or gastralgia and pyrosis. Constipation and headache are the more common symptoms." This is the therapeutical effect of iron used as a medicine. When larger doses are used, or doses which produce the last-named effects are continued in spite of the constitutional disturbances, the poisonous properties become pronounced, or, in other words, the exaggerated therapeutical effects are observed, and the patient or victim will suffer from an intense inflammatory action of the whole alimentary tract, and will suffer from the symptoms of a gastrointestinal irritation, marked by pains similar to those which have been described under the head of irritants,—pain, nausea, vomiting, diarrhea, and serious constitutional disturbance. These symptoms will follow inordinate doses of any preparation of iron, the most irritant of them causing

[96] Edes' Therapeutic Handbook of the U. S. Pharmacopœia, N. Y. p. 126.

the symptoms from smaller doses, the less irritant from larger doses. The cases in which medicinal iron preparations would be used with suicidal or criminal intent are naturally rare.

314. Symptoms of sulphate of iron poisoning.— A case of supposed criminal poisoning with this substance is related by Dr. Christison. A girl, four years of age and previously in good health, was attacked with violent vomiting and purging immediately after breakfasting on porridge, and died in the course of the afternoon of the same day. The porridge had a blue color, and it was proved that a woman in the house had purchased both this salt and the sulphate of copper. The body being disinterred four months after death, the stomach was found soft, gelatinous, and of a uniform, intense black color through the whole thickness of its parietes, and the entire alimentary canal lined with a thick layer of jet black mucus "from the pharynx down to the very anus." There was no evidence found of the presence of copper, but abundant proof was obtained of the presence of iron, both in the textures of the stomach and the black mucus which lined it.[97] More recently, a case somewhat similar has been observed by Orfila. It was that of a child, aged fifteen months, who died after purging and vomiting a black fluid. On opening the body ten days after burial, the stomach was found to be filled with a greenish fluid, and the vessels of the lungs and brain were gorged with black blood. M. Orfila detected sulphate of iron, in notable quantities, in the portions of the abdominal contents forwarded to him.

315. Symptoms of chlorid of iron poisoning.— The medicinal tincture of this salt of iron has frequently given rise to serious and fatal accidents. The symptoms produced by it are very much like those of the corrosive acids, *viz.*, heat, dryness, and swelling of the throat, with a burning pain in the stomach and in the course of the œsophagus, vomiting of blood, and inky evacuations. Its corrosive properties seem to be due to the presence in it of free hydrochloric acid. Dr. Christison relates a case in which death occurred in about six weeks after 1½ ounces (46.65 C.C.) of the tincture had been swallowed. A case of recovery after 3 ounces (93.30 gm.) of the concentrated tincture had been swallowed is reported by Sir William Murray.[98]

A gentleman, aged seventy-two, swallowed 3 ounces of it by mistake. He was found "tossing about in the utmost consternation and agony; his tongue was swollen, and protruded from the mouth; its skin was parched and peeling off, while ropy mucus flowed from the mouth and nose; the eyes seemed starting from their sockets; the

[97] Pereira, Mat. Med. p. 393. [98] Am. Jour. of Med. Sci. July, 1849.

respiration was noisy and laborious, and suffocation seemed to be impending. During this time his hand was riveted to the region of the stomach, as the principal seat of pain; the palate and the interior of the mouth were burned, and presented a parboiled appearance." The acid was first neutralized by an alkaline mixture, and this treatment was followed by demulcents and laxatives. The gentleman rapidly recovered.[99] Several other cases of recovery from large doses are recorded, which it is not necessary to particularize.

The tincture of the chlorid of iron, as it is now called, contains a notable amount of free hydrochloric acid,—especially when it has been recently prepared,—and hence poisoning by this substance is similar to that of weak hydrochloric acid, intensified by the irritant action of the chlorid of iron.

One case of suicide is recorded by Mr. Blyth as having occurred in England during his ten years collation of deaths by poisoning. He also quotes a case where the tincture of the chlorid of iron was used in Martinique[100] to poison no less than four different persons. This drug was administered in a glass of punch to the victims, on an empty stomach; they had been drinking spirits, but had eaten nothing solid from noon until eight o'clock in the evening. One of the victims died after about thirteen hours' illness; two died after sixty-five hours' illness; the fourth lived from three to four days. In the first victim, the symptoms were very pronounced in an hour; these were violent, colicky pains in the abdomen, associated with diarrhea, but there was no vomiting; the second and third cases suffered from abdominal pains and suppression of the urine; the fourth case showed violent, abdominal pains, there was constipation and suppression of urine; he also suffered from painful tenesmus. The amount of the poison was not stated.

Perchlorid of iron in solution has not unfrequently been used as a hemostatic, locally applied to the interior of the uterus; the local irritation has occurred, and even been followed by violent inflammation, succeeded by peritonitis and death.

316. Symptoms of subacute and chronic poisoning.— These are not dangerous, and to a well-educated physician would be very apparent, and be relieved by discontinuance of the iron preparation.

317. Lethal dose.— This is difficult to determine, from the small number of fatal cases which have been reported. Serious symptoms have arisen after 1 ounce (31.19 gm.) of the chlorid of iron, and one

[99] Sir Wm. Murray, Dub. Med. Press, Feb. 1849.

[100] Bérenger-Féraud et Porte in Ann. d'Hygiène Publique, 1879.

death has been reported from the solution of chlorid salt equal to 1.5 ounces (46.65 C.C.) of the tincture of chlorid of iron; our case, above quoted in the preceding paragraph of a gentleman aged seventy-two, shows that, after appropriate treatment, an amount of 3 ounces does not produce fatal results.

318. Treatment.— Carbonate of soda in weak solution should be immediately given, and pumped out of the stomach; milk and emollient drinks should then be freely given until the intestines have been cleared of the iron salt. This will be made apparent by the change of black color in the stools to brown. Appropriate medication should be given to relieve pain.

319. Action.— Bérenger-Férraud et Porte have made some very elaborate experiments on rabbits, dogs, and cats, with chlorid of iron both (anhydrous and aqueous solution of ferric chlorid). The first series of experiments comprises the administration of the solution of chlorid of iron, enclosed in gelatine capsules, with the food of the animal. No symptoms except of a trifling character followed its ingestion even with doses exceeding one gram per kilo weight of the animal; the anhydrous chlorid of iron, as also the solution, were directly introduced into the stomach, without causing fatal poisoning, and the effects soon subsided. Quantities of the solution in doses of .5 of a gram per kilo weight of the animal were administered to animals on a fasting stomach; with this dose, and even larger doses, death followed in from one to sixteen hours, the poisonous action being increased, and the period of death shortened, by the addition of alcohol; the ordinary tincture of the chlorid of iron was used in these cases; the symptoms of vomiting (although sometimes absent) and diarrhea occurred; in a few instances there was paralysis of the hind legs, dilation of the pupils, and scanty (sometimes complete suppression) of urine; convulsions preceded death.

320. Elimination of iron chlorid.— This is accomplished by its conversion into a sulphid, and its expulsion with the feces by the intestines, the stools being black; a smaller portion is excreted by the kidneys in the urine. Iron, existing in an abnormal amount in the circulation of the body, passing through the renal circulation, apparently produces, as in the case of other metals, an irritation and inflammation of the lining membranes of the tubules of the kidneys (parenchymatous nephritis). This action, as well as the changes previously described, in arresting the functions of the liver secretion, will explain the therapeutical action of absorbed iron in inordinate doses,

as especially noted in the introductory remarks on iron in the first part of this article.

321. Diagnosis.— We should expect that the educated physician would recognize symptoms of precordial distress and disturbed digestion, occurring in a feeble patient after excessive doses of iron, as being due to an overdose. Unless the ferruginous treatment is discontinued, the more aggravating symptoms of gastric pain, excessive constipation followed by diarrhea, the especial pressure experienced in the head, and albuminuria, would supervene.

322. Post-mortem appearances.—In one of the cases, referred to as happening at Martinique, the details of the autopsy are recorded as follows: Tongue covered with a greenish fur, bordered at the edges with a black substance, described as being like "mud;" the lining membrane of the gullet was pale, but also covered with this dark "mud;" the stomach contained a greenish-black liquid; the liver was large and congested; the kidneys were swollen, congested, and ecchymosed; the cerebral membranes were gorged with blood, and the whole brain hyperemic. In the experiments on animals, previously referred to, the general changes noted were dryness, pallor, and parchment-like appearance of the cavity of the mouth, the mucous membrane being blackened by contact with the liquid. When the dose was large and concentrated, there occurred one or more hyperemic spots on the mucous membrane, but otherwise this membrane had a healthy look. The interior of the intestines showed no inflammation, and, on exposure to the air, the brownish coloring of the mucous surface became black. The conditions of the liver, kidneys, membranes of the brain, and the brain substance itself, were similar to the appearances observed in the case of the victim at Martinique, whose autopsy is above related.

323. Detection and tests.— It should not be forgotten, in making a chemical examination of the viscera, in cases of supposed poisoning with the salts of iron, that iron is a normal constituent of the body and food. The saliva contains traces, according to Prof. Bernard, Besanez, Wright and Enderlin, Humbert and Lassaigne. The intestinal secretions, as well as the gastric juice and the bile, also contain a considerable amount of iron, which comes mainly from the food. A man of average weight secretes about 18 grains a day with the gastric juice, and about 20 grains with the bile during the same period. In fact, all the secretions and excretions from the body contain iron varying in amount from a trace to several grains, so that 2½ per cent of the ashes of the burned body are oxid of iron. More-

over, that all the iron of the body does not come from food is shown by a well-known, physiological experiment of feeding dogs on food almost deprived of iron, where it is shown that the elimination of iron far outweighs its absorption. A dog, which was fed for twenty-seven days on food containing a total amount of 39.5 milligrams, eliminated 89.5 milligrams, and afterwards received 116 milligrams, and excreted 144.5 milligrams. The blood in man contains by estimation a little more than ½ per cent of iron by weight, and a man of average weight would have in his blood about 3 grams, or 40 to 50 grains of iron. On account, therefore, of the large amount of normal iron in the animal economy, it is necessary in legal analyses to be able to isolate a very large amount, much more than the normal. Several criminal cases have been investigated in France,[1] so that such analyses are sometimes necessary; but usually such large doses have been taken in these cases that large amounts remain in the stomach or intestines adherent to the lining membrane, either changed into the form of sulphid, which imparts a black color to the membrane, or unchanged, if death has taken place very rapidly. If the iron exists in the contents of the stomach or intestines in the form of sulphid or sulphate of iron, it can be easily separated by treating with hydrochloric acid, straining and filtering, when the filtrate will contain the iron which can be separated by boiling with nitric acid to convert it into the form of ferric chlorid, and precipitating, by the addition of ammonia water in the form of ferric hydrate, a reddish-brown precipitate. This, after being washed, can be dissolved in hydrochloric acid and the solution of chlorid of iron tested; (1) by adding a solution of sulphocyanid of potassium, which gives a blood-red solution of sulphocyanid of iron, which is not bleached by the addition of free mineral acids nor by a solution of corrosive sublimate; (2) by neutralizing or rendering slightly alkaline with sodium hydrate, adding a solution of ferrocyanid of potassium and then acidulating with hydrochloric acid, when a dark blue precipitate will be formed (Prussian blue); and (3) by rendering alkaline with ammonia water, which will precipitate the hydrate of iron, and then adding sulphid of ammonium, which will change the hydrate of iron to the form of the black sulphid of iron.

XXIV. Zinc.

324. Oxid of zinc, in general.—Oxid of zinc has been, of late years,

[1] Journal de Chimie Médicale, 1874, p. 149.

used as a substitute for white lead, with the view of avoiding the dangerous effects of the latter on the workmen. It has been supposed to be innocuous, and this idea appeared to be confirmed by some experiments made by M. Flandin. He rubbed animals over with ointments of oxid of zinc, of carbonate of lead, and of sulphate of lead; the last two were found always to produce poisonous effects, but the animals, rubbed with the ointment of oxid of zinc continued to enjoy their usual health. A case has, however, been recorded by Dr. Bouvier, of the Hôpital Beaujon, at Paris, in which a laborer, who had been employed for fifteen days in barreling oxid of zinc, and who, in other ways, had handled this substance, and breathed an atmosphere loaded with its powder, was attacked with vomiting, colic, and constipation. These symptoms persisted, and increased in intensity so much that he rolled on the floor in agony. The vomited matters were bilious, he rejected his food almost immediately after swallowing it, and he had been constipated for five days. From the whole history of the case, it was considered to be one of genuine zinc colic. He was cured by the remedies usually employed for painter's colic. The particles adhering to his body were examined, and found to consist of oxid of zinc.[2] Ladouzy and Maumené have seen workmen, who were obliged to inhale an atmosphere loaded with particles of oxid of zinc, affected with inflammation of the mouth and throat, salivation, general distress, colic, and diarrhea, or obstinate constipation.[3]

It must not be forgotten that oxid of zinc is very likely to be contaminated with salts of lead and arsenic, and, as many of the above symptoms are those of poisoning by either of these metals, it is highly probable that the suspected case was not due to the zinc oxid. It should, at least, be shown in evidence that no such contamination was possible in a given case of suspicion.

325. Symptoms of oxid of zinc poisoning.— Cases of zinc poisoning, arising from the inhalation of the oxid of zinc, have also been observed among the workmen engaged in twisting and beating the iron wires galvanized with zinc used for securing champagne corks. Four had symptoms of general depression, with sore throat, swelling and ulceration of the tonsils, salivation, fetid breath, colic, and diarrhea. In one case there were colic and obstinate constipation. These symptoms subsided readily on abandoning the occupation, and did not return when the work people resumed their work with wires better pre-

[2] Am. Jour. of Med. Sci. Oct. 1850, from the Comptes Rendus.

[3] Briand, Méd. Lég. 6ème ed., p. 433.

pared, and free from loose oxid or carbonate of zinc.[4] M. Blandet has described, as effects of breathing the vapors of zinc fused at the temperature required to melt copper, chilliness, trembling, headache, fainting, vomiting, buzzing in the ears, contusive muscular pains, etc.[5] In these cases it is more probable that the galvanized iron contained impurities of lead, than that zinc oxid was the cause of the poisonous symptoms.

326. **Poisonous nature of zinc.**—With regard to zinc as a poison, the best authorities are in disagreement. Briand and Chaudé, in their excellent work, declare that metallic zinc is not poisonous.[6] If accidents have occurred to workmen who work with this metal, they affirm that these must be due to the presence of arsenic in the zinc. Dr. Taylor[7] mentions the case of an epileptic, who took, in the course of seven months, 1 pound, having taken in one day 70 grains. Tardieu does not mention zinc as a poison in his work on toxicology. Neither the oxid nor the sulphate of zinc can be regarded as powerful irritants. There is no case, we believe, on record where this agent has been proved incontestably to have caused death. Dr. Schockow[8] gives symptoms of poisoning by those who work in zinc mines; but, as these symptoms are similar to those of arsenic and lead, and as these metals are present in the metallic ore used in the smelting furnaces, his evidence is of small account, as compared with the statements of toxicologists of higher authority, who have been concerned with the history of persons who have taken large medicinal doses without accident. These remarks apply equally to the so-called cases of chronic poisoning by zinc oxid.

The chlorid and sulphate of zinc, being constituents of soldering fluids, and containing free hydrochloric acid, are easily obtainable; and 11 cases of fatal poisoning by these salts of zinc are reported by Mr. Blyth, 7 of which were by accident, and 4 by suicide. In the table of the medical examiners' returns for Massachusetts (page 20) there are reported 10 fatal cases of poisoning by these salts, all suicidal. Witthaus and Becker have collected 55 cases of fatal poisoning caused by these two salts, of which 46 were in Great Britain, and only 3 in the United States. Sulphate of zinc was used in 25 cases, in 8 of which it was taken by mistake for Epsom salts (sulphate of magnesia), 5 were suicidal, and 4 were homicidal. Of the other 40 cases,

[4] Am. Jour. Med. Sci. Oct. 1850, from Monthly Jour. Aug. 1850.
[5] Jour. de Méd. 1845, p. 76.
[6] *Ibid.* p. 672.
[7] Medical Jurisprudence, p. 255.
[8] Deutsches med. Wochensch. 1879, 208, from H. C. Wood's Mat. Med. and Toxicology, Phila. 1882.

27 were accidental, and 7 suicidal. In 26 of these cases Burnett's disinfectant was used in soldering fluid.

327. Sulphate of zinc, in general.—Sulphate of zinc, white vitriol. $ZnSO_4 + 7H_2O$, is a white, crystalline solid, freely soluble in water, slightly soluble in alcohol. Its solutions have an acid reaction and a strong, astringent taste. It is used extensively in medicine as an emetic and as an external astringent.

The prompt emetic action of sulphate of zinc is the cause of its seldom producing serious effects. The dose usually administered with a view to its emetic operation is from 15 grains to ½ dram (1 to 2 gm.) and serious results have seldom been witnessed from this. Dr. Babington once gave 36 grains three times a day, for several weeks, without any sickness or other untoward effect being produced; but cases in which the stomach would tolerate such doses as these must be very rare.[9] It should be remembered that the sulphate of zinc is very commonly used as an emetic in cases where the physician wishes to get rid of poisons from the stomach, and that it is never in his mind that he is giving another poison when he administers this drug. The records and expérience of the medical profession should outweigh all other doubtful evidence.

Christison mentions several cases in which the salts of zinc were supposed to have been the cause of death; yet some of these seemed to him doubtful. "Even in large doses it can hardly be accounted poisonous, as it merely gives rise to vomiting and slight diarrhea; and that an adulteration to such an amount would always betray itself by its strong disagreeable taste."[10] The best-marked cases are, however, those which have been reported as occurring at Pavia. The first case occurred in the person of a strong woman, who took, by mistake for Epsom salts, a solution of 1½ ounces (46.65 gm.) of sulphate of zinc. She instantly vomited, and then became affected with almost incessant retching and purging for half an hour, which continued afterwards, at short intervals, for three hours, and then gradually diminished. The pulse was frequent and small, and extreme prostration existed, accompanied with distressing restlessness and anxiety; the temperature of the skin was diminished; great pain in the abdomen, limbs, etc., existed, as well as a sense of burning in the throat and stomach. She died thirteen and a half hours after taking the poison, retaining her intellectual faculties to the last.

[9] Guy's Hosp. Rep. Vol. XII. p. 17.
[10] Christison, op. cit. p. 450; and Orfila's Toxicologie, I. 567.

328. Post-mortem appearances after poisoning by sulphate of zinc.—On examination, forty hours after death, the following were the chief appearances observed: Great lividity of the skin; congestion of the brain and its membranes; congestion of the lungs; flaccidity of the heart; the inner surface of the stomach covered with a yellowish, pultaceous matter, on the removal of which a uniform, yellow, ocherous color was observed, except towards the great curvature, where it became reddish; a gelatiniform softening (*ramollissement*) of the mucous membrane prevailed, exposing in some parts the submucous cellular tissue. The small intestine was somewhat injected, and contained yellowish matters. In the second case a similar dose was taken, followed by nearly the same symptoms, but the patient recovered. In the third case ¼ of an ounce (7.77 gm.) was taken, which produced the same symptoms of irritant poisoning, ending in recovery. In the fourth case, of which little account is furnished, but which proved fatal, it is stated that "2 drams (7.77 gm.) of sulphate of zinc were detected in the liver and blood, the fluids of the alimentary canal furnishing but little."[11]

One case, in which the sulphate of zinc was supposed to have been given with criminal intentions, became the subject of judicial inquiry in France. An old man died somewhat suddenly, having suffered from severe pain and great heat in the chest and abdomen, with violent vomiting and purging. He was not seen by a physician. On inspection, the stomach and bowels were found highly inflamed, and sulphate of zinc was found in the contents of the stomach, and detected in the tissues. The body of a woman, who had died two months previously, was also disinterred, and sulphate of zinc found in the viscera.[12] Violent enteritis was also observed in a case reported by Krauss.[13] Dr. Gibb has reported the case of a lady who took, by mistake, about 67 grains of sulphate of zinc in solution. She recovered, and her more serious symptoms were probably owing to 2 grains (0.13 gm.) of tartar emetic ignorantly given to her.[14] A case has been recorded by Dr. Ogle of a drunkard who attempted to commit suicide by cutting his throat. It was believed, but upon no direct evidence, that he had been in the habit of swallowing a strong lotion which he was using for inflamed eyes. The immediate cause of death was not determined; but the report states that an examination of the body revealed the presence of sulphate of zinc in the stomach, a white and

[11] Brit. and For. Med.-Chir. Rev. April, 1849.

[12] Jour. de Chimie Méd. 1845, p. 529.

[13] Canstatt's Jahresbericht, 1853.

[14] Lancet, May, 1850, p. 540.

shriveled appearance of the mouth and fauces, a condensed, indurated, and tripe-like appearance of the lining membrane of the stomach, and to some degree of the small intestine, and an unusually contracted state of the colon and rectum.[15] Two more recent cases are recorded by Dr. Niemann.[16] In the first, a sickly man died with violent, gastric pains and vomiting, after a dose administered to him by his wife; and sulphate of zinc was found, upon chemical analysis, in the contents of the stomach. The second case was one of suicide. In neither was the quantity of the poison determined.

329. Detection and tests of sulphate of zinc poisoning.—From its solution, if pure, the oxid is thrown down by the caustic alkalies, in the form of a white hydrate, which is easily soluble in an excess of the precipitant. Sulphid of ammonium gives a white precipitate, and also sulphureted hydrogen, provided there is no free acid in the solution. Carbonate of ammonium precipitates carbonate of zinc, also white, which is readily dissolved in an excess of the precipitant. Ferrocyanid of potassium also causes a white precipitate. If mixed with carbonate of sodium, and exposed to the action of the reducing or inner flame of the blowpipe upon charcoal, the charcoal becomes covered with a coating of oxid of zinc, which is yellow while hot, but becomes white upon cooling. If a compound of zinc be moistened with a solution of nitrate of cobalt, and heated by the blowpipe, a mass is left which has a beautiful green color. The sulphid of ammonium is the most characteristic and unobjectionable of these reagents; for zinc is the only metal, with the exception of aluminium, whose salts are thrown down white by it, but zinc can easily be distinguished because, though the aluminium salts are also thrown down, the precipitate is not soluble in an excess of the reagent. Having discovered the base, the presence of sulphuric acid in the combination may be easily detected, by testing with chlorid of barium.

330. Chlorid of zinc, in general.—Chlorid of zinc, a very soluble, deliquescent salt, is a constituent of Burnett's disinfectant, and in the "soft injection" fluid used for preserving bodies for dissection.

331. Symptoms of chlorid of zinc poisoning.— The external use of chlorid of zinc, applied locally to a cancerous breast (Blyth) by a quack at Barnstaple, Devon, England, was followed by general symptoms of internal poisoning by zinc, and that metal was found in the liver and other organs. The symptoms of fatal poisoning by chlorid of zinc are immediate pain in the throat, burning of the lips, tongue,

[15] *Ibid.* Aug. 1859, p. 210. [16] Henke's Zeitschrift, LXXVIII. 219.

etc., difficulty in swallowing, increase of saliva, bloody vomiting, diarrhea, collapse, coma, and death sometimes within a few hours.

332. Symptoms of subacute poisoning by chlorid of zinc.— Where death has been delayed beyond the third day, there is gastrointestinal irritation, followed later by diarrhea; after the lapse of a fortnight, perversion of taste and smell, aphonia, spasms of muscles, muscular weakness, and impaired sight,—all referable to inhibition of nervous centers. Other symptoms are a catarrhal condition of the stomach and intestines, chills with pains in the back, pains in the forehead, sweating, and gradual convalescence. The local inflammatory action upon the throat may result in stricture of the gullet and of the pylorus, and, also, destruction of the stomach glands, the results of which would be inanition from impaired digestion, and death.

333. Post-mortem appearances after poisoning by chlorid of zinc.— These vary according to periods at which death took place. After acute poisoning, the lining membrane of the mouth and gullet is white and opaque, of the stomach hard and leathery, shriveled, corrugated, or ulcerated.

After subacute and chronic poisoning, constriction of the gullet and of the cardiac portions of the stomach may be present, and show similar lesions as described under the head of mineral acids. In these cases it would be a question whether the inflammatory processes were not more likely due to free hydrochloric acid contained in the soldering solution containing chlorid of zinc. In one of Dr. Markham's cases, in a patient who died ten weeks after the ingestion of chlorid of zinc, there was noticed a constriction at the pyloric end of the stomach, so that a quill could hardly pass through the stricture. In another case (Blyth) there is preserved in Guy's Hospital a preparation of the stomach of a patient who died fourteen weeks after taking a tablespoonful of Burnett's disinfectant: This stomach, much contracted (to 5 inches in length), has ulcerations both at the pylorus and at the entrance of the gullet; near the cardiac portion of the stomach is a pouch-like dilatation of the mucous membrane, adherent to the spleen; this communicates by a perforation with an abscess, whose limits are bounded by the stomach, diaphragm, and spleen, and originally contained 3 ounces of dirty-looking pus. At the pyloric orifice there is a second perforation, but the contents were confined by the adherent omentum and transverse colon. The muscular coats appeared thickened.

XXV. Tin.

334. In general.—Tin is used in the solder in canned meats, fruits, and vegetables. It is doubtful if this can serve as a source of poisoning; for Roessing[16a] claims that tin is not acted upon by the digestive juices.

335. Chlorid of tin.— This preparation needs little notice: it is an irritant poison, but has seldom given rise to accidents. An old man dried some wet, cooking salt in a tin dish upon a stove, and then ate some meat and bread with which he had wiped the dish. He was seized with chilliness, violent pain in the stomach, and the abdomen became swollen and tender upon pressure. A febrile condition was soon set up; but the most striking symptom was salivation, with extreme fetor of the breath, and a grayish discoloration of the gums. They, as well as the tongue and inside of the cheeks, became covered with ulcers. By an appropriate treatment and gargles of chlorid of lime, he was restored in a few days.[17]

336. Poisonous nature of tin and its salts, in general.—It is highly improbable that tin and its salts are poisonous *per se,* but on account of other metallic contaminations; and the case above cited is evidently illustrative of this theory, since the tin dish, so called, could hardly be other than an iron dish plated with tin. The plating of tin, either on copper or iron, is well known not to be pure tin plate, the tin of which is insoluble, or without irritant or corrosive properties. Among the contaminations of tinned metal is, principally, lead, which, on account of its cheaper price, is largely used in place of tin. The proportion of lead to the amalgam, used to coat copper and iron, varies from 10 to 50 per cent; and when the amalgam coating contains the latter amount, it is easily dissolved in water or the weak, organic acids, and hence gives rise to symptoms of lead poisoning in those who may be exposed to its absorption. The large amount of lead used in the tin-lead amalgam causes a bluish-white appearance, and hence artisans combine a certain amount of zinc to whiten this coating, and to give it a bright, white luster; the amount of zinc added to the amalgam varies from 15 to 25 per cent. Consequently, who would dare to impute the well-known symptoms above related as peculiar to tin poisoning, when the amount of tin used in tin plate is reduced to 25 per cent for the least, or 75 for the heaviest plated ware? Is it not more reasonable, unless chemical research shall dis-

[16a] Zeitschr. f. allg. Chem. 1900.
[17] Memel. Deutsche Klinik, No. XII. 1851.

prove the fact, to attribute these symptoms to poisoning by lead, and in the case above quoted, to chlorid of lead (from decomposition of the cooking salt), which is the most active and dangerous of lead salts? To those of our readers who desire to know more of this dangerous, because so extensive, use of lead, reference is made to the article Etain,[18] and to the bibliographical references therein contained.

Since the above was written in the fourth edition of this work, there have occurred a few cases where toxic symptoms followed the ingestion of tin salts. A chemist swallowed, by mistake, in some putty powder, a higher oxid of tin; he had used it for months in his pepper box.[19] Luff[20] mentions four cases of poisoning by tin, in a tin can of cherries, in which it was estimated that 1.9 grains (0.123 gm.) of the higher oxid of tin were contained in each fluidounce (about 30 c.c.) of the cherry juice contained in the can. This would be equal to 3.2 grains (0.207 gm.) of the malate of tin in the same quantity of juice. The danger of tin salts poisoning, in cans containing preserved meats, is less likely to happen, because the amount is never large in the solder used for sealing them.

337. Symptoms of poisoning.—These, in the four cases mentioned by Dr. Luff, were very severe,—nausea accompanied with a strong, metallic taste; abdominal pains followed by vomiting and diarrhea; symptoms of collapse, with feeble, irregular, and rapid pulse; and occasionally, cyanosis, collapse, unconsciousness (in one case), muscular cramps, and albuminuria. All these four cases recovered. The case of the chemist, above quoted, was fatal, after an illness of a couple of months.

According to Unjar and Bodlander, experiments upon the lower animals to which were given hypodermic doses of nonirritating tin salts, produced poisonous symptoms like those of the other metals, and undermined their health, sometimes even causing death; tin was found in the tissues and urine of these animals.

338. Lethal dose.— This, on account of the few cases of fatal poisoning recorded, cannot be stated with any degree of precision.

339. Treatment.—The stomach should be emptied by a mild emetic (not sulphate of zinc) of mustard and water, or by the stomach pump. The collapse should be treated by alcoholic stimulants, and the pain by opium; demulcent drinks freely administered.

[18] Nouveau Dict. de Méd. et de Chir. tome XIV. p. 278 *et seq.*

[19] Med. Press and Circ. 1894.

[20] Luff, op. cit. Vol. I. p. 213.

340. Post-mortem appearances.—There are none recorded.

341. Tests.—Tin forms two kinds of salts,—stannous and stannic. The stannous salts give the following reactions: With hydrogen sulphid, a brown precipitate of stannous sulphid; with mercury chlorid, a gray deposit of mercury; with fixed alkalies, a white precipitate, soluble in excess of reagent and reprecipitated on boiling. The stannic salts give the following reactions: With hydrogen sulphid, a yellow precipitate of stannic sulphid; with fixed alkalies, a white precipitate, soluble in excess of reagent, but not reprecipitated by boiling.

The method usually adopted is that used for the separation of arsenic from organic tissues, as already described. After removal of the organic sulphid, the tin, if present, will be in the form of a stannic salt, and can be determined by the above tests.

XXVI. Silver, gold, and platinum.

342. Nitrate of silver, in general.—Silver nitrate, $AgNO_3$, lunar caustic, crystallizes in colorless, rhombic plates, very soluble in water, with a metallic taste. In medicine it is used principally as an escharotic.

343. Cases of poisoning by nitrate of silver.— Poisoning by it is not, however, of frequent occurrence. A patient at the Hôpital St. Louis, in Paris, recovered, after having swallowed 1 ounce in solution. The nitrate of silver was neutralized by the administration of common salt.[21] In a case reported by Krahmer, a like quantity produced insensibility to tactile impressions, loss of consciousness, and convulsions. By the sixth day the patient had recovered.[22] The nature of the poison can be roughly detected by the grayish color which it communicates to organic matter.

Witthaus and Becker have collected 7 cases of poisoning by nitrate of silver, which were accidental, from swallowing this caustic when used in the mouth or throat for local affections; 5 were children (one fatal case), and 2 were adults.

344. Preparations containing nitrate of silver.— There are several preparations containing nitrate of silver, among which may be noted the following: (U. S. P.) (Mitigated caustic) Diluted silver nitrate, made by the fusing of 30 parts of silver nitrate with 60 parts of

[21] Am. Jour. 1840, p. 239.
[22] Canstatt's Jahresbericht, 1846, p. 247.

potassium nitrate; silver oxid as a medicine; silver iodid also as a medicine.

345. **Hair dyes.**— Aqua orientalis, containing of grain silver 2 drams, of nitric acid 1 ounce, some steel filings, distilled water 1½ ounces, water added to make 3½ ounces, and filtered. (Argentan tincture) Nitrate of silver 1 dram, rose water 1 fluidounce, sufficient nitrate of copper to impart a greenish tint. (Eau d'Afrique) Two solutions,—one of nitrate of silver, the other of potash, containing ammonium sulphid.

346. **Marking inks.**— Indelible inks contain varying quantities of silver nitrate from 8 per cent to 30 per cent.

347. **Symptoms of acute poisoning.**— White eschars in mouth; griping pains; vomiting of light-colored masses, turning black when exposed to light; occasionally the vomited matters may contain blood; purging diarrhea generally occurs with the appearance of blood in the stools; collapse, muscular cramps, unconsciousness.

A case is reported of fatal poisoning by a piece of silver nitrate (¾ of an inch long) having fallen down the throat of a child fifteen months old; vomiting immediately occurred, followed by convulsions and diarrhea; sodium chlorid was given, but the child died in six hours.[23] In other cases paralysis and unconsciousness have been reported.

348. **Subacute and chronic poisoning.**— Silver salts in the form of nitrate and iodid have frequently been prescribed for a long time in chronic nervous diseases. In these cases a peculiar bluish complexion will follow, probably due to the deposit of finely divided granules of metallic silver in the skin. This effect of patches of a similar hue is more marked in those parts of the body exposed to the sunlight; yet the color is not confined to the surface of skin, but is also seen in the mucous membrane, showing a violet line around the edge of the gums. This effect has been observed after a two months' continuous treatment of a patient with silver nitrate, the whole quantity taken being 60 grains (a little less than 4 gm.) Riemer records a case (Blyth) in which, after a year's use of a total quantity of 17.4 grams, a grayish-black color of the face was produced; later, when nearly double the quantity had been taken, the color invaded the whole body. This staining of the skin begins first around the nails and fingers. The administration of small doses of silver nitrate appears to inhibit the function of a reflex action centered in the central nervous system.

[23] Brit. Med. Jour. May, 1871.

349. Lethal dose.—50 grains (about 3.2 gm.) has proved fatal to an adult. The administration of ½ grain to 2 grains (.03 to .13 gm.) of the silver nitrate, and the same quantities of the silver oxid is used continuously as medicinal treatment.

350. Treatment in acute cases.—Chlorid of sodium (table salt), albuminous drinks, and milk. Where, occasionally, chronic poisoning may have occurred, as, for instance, with ulceration of the stomach, and the discoloration of skin, hair follicles, perspiration glands, also of the lips and of the gums, there is no antidote.

351. Action.—Silver, as well as the other metals, iron, nickel, cobalt, and manganese, and other heavy metals, pass exclusively from the blood into the intestines, being secreted by means of Lieberkühn's glands; if there is a large quantity of these in the blood they are likely to be excreted by the kidneys, and cause parenchymatous nephritis. The main quantity of the metal circulating with the blood is held by the liver, whence it is slowly transported toward the intestines by leucocytes.

352. Diagnosis.—In acute cases, by appearance of the vomit above named, light-colored masses, turning black when exposed to light; white eschars in mouth.

353. Post-mortem appearances.—In acute cases, gastroenteritis. In chronic cases, ulceration of the œsophagus and stomach, blackening of the glomeruli of the kidneys, Glisson's capsule of the liver, the villi of the small intestines, and medullary portion of the bones. Sections of the blackened organs can be seen to be decolorized under the microscope, by cyanid of potassium.

354. Tests and detection.— Soluble salts of silver, when added to a solution of hydrochloric acid or soluble chlorid, precipitate chlorid of silver, as a white, curdy mass, soluble in ammonia, but insoluble in nitric acid; with potassium iodid, soluble salts of silver give a yellowish-white precipitate of silver iodid, insoluble in nitric acid, and only very slightly soluble in ammonia; with potassium chromate, a red precipitate; with sulphureted hydrogen, a brownish-black precipitate, insoluble in ammonium sulphohydrate; with KOH or NaOH, a brown precipitate, insoluble in excess, but soluble in ammonia.

In the destruction of organic matter, as described under arsenic, the silver is precipitated in the form of silver chlorid. This can be collected on a filter, washed, dissolved in ammonium hydrate, reprecipitated by hydrochloric acid, etc. After repeating this process of purification once or twice, the identity of the silver can be determined by the above tests. Also a portion of silver chlorid precipitate, after being purified, can be reduced on charcoal to metallic silver.

355. Trichlorid of gold.—The trichlorid of gold is also a highly irritant poison, acting very much like corrosive sublimate. Cullerier, the nephew, has seen 1/15 of a grain (.0044 gm.) excite, at the second dose, gastric irritation, dryness of the tongue, redness of the throat, colic, and diarrhea.[24]

356. Bichlorid of platinum.—The bichlorid of platinum is a powerful, caustic poison. It is rarely used, in medical practice, for the treatment of secondary syphilis.

XXVII. Neutral sodium, potassium, and ammonium salts.

357. Action of salts of sodium, potassium, and ammonium, in general.—The neutral salts of sodium, potassium, and ammonium, formed by the union of the sulphuric acid, hydrochloric acid, tartaric acid, citric acid, and in combination with halogens, bromin, and iodin, produce a poisonous effect, on account of the alkaline metal, rather than from the acid, in combination. The poisonous action of the potassium salts is the strongest, and that of the sodium salts is the weakest, while that of the ammonium salts lies between these two. If the poisonous action of the sodium salt be placed at 1, the ammonium salt might be considered twice as powerful, or at 2, and the potassium salt three times as powerful, or at 3. These calculations have been based by the action of these neutral salts upon the central organs of circulation and of respiration, yet the indications appear to show that their relative action upon the whole organism is in similar proportions as upon the two center organs above indicated. While this statement is based mainly upon experiments upon animals, yet the results of clinical experience seem to corroborate this statement. Some of the fatal cases of poisoning by these neutral salts, few in number, date back to the period of 1837 to 1856. The experimental study of their action is of a much later period. There may be one exception to this theory, in the case of alkaline salts of iodin. Kobert proffers the theory that in this case the iodin may be liberated within the organism, possibly by the nitrous acid contained in the mucus of the nose, or by some yet unknown action of enzymes from the fungi of suppuration. This writer classifies enzymes as pepsin, trypsin, ptyalin, diastase, emulsin, and myrosin. Kobert also calls attention to the fact that these alkaline salts may, in concentrated solution, produce an entirely different poisonous action than when given in the ordinary solution used in disease by physicians. The concentrated

[24] Pereira, Mat. Med.

solution, even including table salt, sodium sulphate (Glauber's salt), and magnesium sulphate (Epsom salts), may produce fatal symptoms of poisoning of a corrosive character, when brought in contact with the mucous membrane. The weaker solutions appear to absorb the watery constituents of the blood, producing a colloidal state of the active cells in the body, while the concentrated solution produces a shriveling and decay of these cells, when coming in contact with them, and also "a reactive inflammation around them." This writer ascribes to the potassium salts a specific exciting action upon the heart and, in larger doses, a paralysis of its muscular walls, while corresponding solutions of the sodium salts are devoid of any such action. The common table salt, in the enormous quantity of half a pound or more, has destroyed human life; the potassic sulphate and its tartrate have destroyed human life.

358. Experiments on animals.—These neutral salts—especially the potassic salt—reduce excitability and contractility of the heart of frogs to a powerful degree; they produce slowing of the pulsations, irregularity, and finally, cessation of its pulsations. When a potassic salt is injected into the blood vessels of a warm-blooded animal, immediate death follows from arrest of the heart's action. The subcutaneous injections of similar doses cause a retardation of the pulse rate, difficult respiration, and convulsions, ending in death; smaller doses still will produce a temporary weakness of arterial pressure, quickly followed by abnormal blood pressure; the first effect, only for a few seconds, increases the rate of pulsation, but, later on, produces a retardation. This effect on the blood pressure in the arteries will occur even after section of the spinal cord. Some of the larger doses of the potassium salts will rapidly diminish the blood pressure, and cause an apparent cessation of the heart's action; but if the thorax be opened, the heart will be seen to contract regularly at about three times the normal rate per minute; if artificial respiration be then maintained, and the walls of the chest be compressed sufficiently to empty the heart of its blood, the arterial blood pressure will increase, and natural respiration may follow. In one of Boehm's experiments, an animal lay apparently dead for thirty-six minutes, but, under the previous method as described, it was brought to life again.

The action of potassium and sodium neutral salts upon the blood is similar; the blood changes to a brighter red, and the form of the corpuscles becomes shriveled through lack of water. The voluntary muscles, into which a solution of potassium neutral salts is injected through the blood vessels, quickly lose their contractility, and the nerves in contact with this solution lose their power of conveying excitability.

The elimination of the neutral salts is by the kidneys, while that of the potassium salts, eliminated through these organs, is accomplished much more slowly; about one fifth (.78 gm.) of a total injection (4 gm.) in solution of potassic chlorid was excreted with the urine in the first sixteen hours, and more than a half of this dose (2.677 gm.) during the following twenty-four hours. (Blyth).

359. Sulphate of potassium in general.— This drug has given rise to quite a number of cases of poisoning, on account of its use by the laity,—especially in France and England, in many puerperal conditions, such as for diminishing the secretion of milk, and for procuring abortion.[25] Some of the accidents caused by its use have been due to impurities which the commercial salt contained, such as the sulphates of zinc and copper, corrosive sublimate, and potassium oxalate and arseniate. Sonnenschein[26] records a case in which a woman took about 10 drams by order of her physician, and died in two hours.

360. Symptoms produced by sulphate of potassium.—The symptoms produced by it are those of an irritant,—severe pain, and burning sensation in the stomach and abdomen; and all of the symptoms of inflammation of the stomach and intestines,—nausea, vomiting, purging, cramps, etc. Taylor[27] records a case of a woman who took a large quantity, presumably 2 ounces, for the purpose of procuring abortion, death taking place after symptoms of severe irritation of the stomach and bowels, and after death, the examination showed evidences of such irritation.

361. Nitrate of potassium in general.— Nitrate of Potassium. (Saltpeter, niter, sal prunella). Potassium nitrate (niter) has been a common remedy in household medicine, and is also largely used as a stable remedy for horses and cattle. It has often been mistaken for Epsom salts and Glauber's salt for use as a purgative. Occasionally, it has been mistaken for table salt, and in one case it was used in a concentrated form, by mistake, as an enema. There are on record 8 cases of poisoning through its mistake for a purgative salt. There are 20 cases of poisoning by potassic nitrate on record.

"A German, who spoke English imperfectly, went into a store, and asked for 'bitter salts,' meaning sulphate of magnesium (*bitter Salz*). The attendant supposed he meant saltpeter, and gave him ½ a pound. The man took 3½ ounces (109 gm.) at one dose. His bowels were opened three times within three or four hours. He com-

[25] Mowbray, London, Med. Gazette, 1869, p. 62. and Ann. d'Hygiène Avril, 1843–44, Vol. XXXIII. p. 54. 1842.

[26] Handbuch der gericht. Chemie.

[27] On Poisons, 1875, p. 265.

plained of a slight sensation of heat in the epigastrium, and drank a good deal of water. About five hours after having taken the saltpeter, he suddenly fell out of his chair and died.

"The marked peculiarity, in this case, was the absence of the painful symptoms which usually follow the ingestion of irritant poisons; and the question, How was death produced? arises. Certainly not by inflammation of the stomach, for he complained of nothing but a slight sense of heat in the stomach. The poison must have acted by destroying the vitality of the blood. There was no post-mortem examination. The rigor mortis was very imperfect, the lips of almost a natural pink hue, and the appearance of the countenance so life-like that some persons who were present doubted the propriety of interment on the third day."[28]

This is the largest dose of niter which we find recorded as having been taken, and its symptoms were, on that account probably, peculiar. There are numerous instances of death occasioned by 1 or 1½ ounces of this salt, in which the symptoms were those of a violent, local irritant, and a depressing action at the same time upon the heart and the nervous power. Death may occur within two hours. On the other hand, recovery has taken place after doses of niter varying from ½ to 2 ounces (15 to 62 gm.). The symptoms are vomiting, with extreme burning pain in the throat and abdomen, followed by coldness and collapse, and, subsequently, by bloody stools. In one case bloody vomiting is noted.[29] Nervous symptoms, such as tremor, spasm, and hallucinations, have been observed. Recovery is slow, and the digestive function remains feeble or deranged.[30]

362. Treatment.— As in the case of the other neutral salts, the stomach should be evacuated by the stomach pump or mild emetics, and washed out. Emollient drinks should then be administered, application of heat to the surface of the body, and stimulants to overcome the feeble action of the heart.

363. Action of nitrate of potassium and of sodium.— The action of the nitrate of potassium and also of sodium is somewhat different

[28] New Jersey Medical Reporter, Jan. 1855.

[29] Jour. f. Pharmakodynamik, II. 178.

[30] Sweet spirits of niter or spirit of nitrous ether, is an alcoholic solution of ethyl nitrate, containing five per cent of the crude ether. There are a few cases of poisoning by large doses, two or three ounces, of this medicine; but in ordinary doses and well diluted it can hardly be called a poison. When it is taken undiluted, it may set up an irritation of the stomach and bowels; but the same may be said of not a few of the regular medicines not ordinarily considered as poisonous. The solution on standing in sunlight a long time will form nitric acid. The few cases reported were among children, or feeble persons (London Lancet, 1878, 2d Pt. 766, and 1879, 1st Pt. p. 9).

from that of the other neutral salts, because, in cases of poisoning from these nitrates, the action of the nitric acid becomes effective. Large doses, 46.3 to 77.2 grains (3 to 5 gm.), of potassic nitrate cause distress in the stomach and pain in the bowels; digestion is also disturbed, and there may be vomiting and diarrhea; there is also irritation of the bladder; a strong desire to urinate frequently. In still larger doses, 231 to 463 grains (15 to 30 gm.), symptoms of acute gastroenteritis, accompanied with great pain and frequent vomiting, are manifested; the vomit may contain blood; there is also irregularity and slowing of the pulse, weakness, cold sweats; painful muscular cramps,—especially in the calves of the legs,—aphonia, convulsions, collapse, and, finally, death supervenes. Unlike the neutral salts first mentioned, pure potassic nitrate does not absorb water. It is believed that the action of the nitrates is due to the double decomposition inside of the body into a nitrate, and with this, more serious irritating action upon the digestive tract is partly due from the free presence of nitric acid, while the nitrates themselves circulate through the blood.

364. Post-mortem appearances after poisoning from nitrate of potassium.—The post-mortem appearances after poisoning from potassic nitrate are inflamed condition of the stomach, darkened mucous membrane, softened, and easily tearing; the contents of the stomach are often mixed with blood. The mucous membrane of the stomach will, in recent cases, be found more or less acted upon by the salt. Thus, it may be seen in some parts reddened, and in others, covered with brown sloughs of partially detached membrane. Perforation has even been observed.

365. Bitartrate of potassium and sulphate of potassium in general.—Potassium bitartrate (cream of tartar) and potassium sulphate have produced fatal action when taken in concentrated large doses. Symptoms were pain in the abdomen, vomiting, purging, syncope, and fatal collapse.

366. Chemical examination of nitrate of potassium.—Nitrate of potassium crystallizes in the form of colorless, prismatic crystals belonging to the rhombic system, and are free from water of crystallization. The crystals decrepitate upon being heated; they are readily soluble in water, but only slightly soluble in alcohol. Its solutions answer to the tests for both potassium and nitric acid. In all cases of suspected poisoning by this substance, an analysis of the urine during life is very important, since it is rapidly eliminated by the kidneys. In examining an organic mixture for nitrate of potassium, it should be evaporated to dryness, extracted with as small a quantity as possi-

ble of boiling water, filtered, and the filtrate tested by the above tests; if necessary, the filtrate can be purified by boiling with pure animal charcoal, and filtering again.

367. Chemical examination of sulphate of potassium.—This salt crystalizes in colorless, hard, rhombic prisms, which have a bitter, saline taste, and are soluble in twelve parts of cold water, but are insoluble in alcohol. It can be isolated by extracting the suspected material with water and filtering; the filtrate should be evaporated to dryness, and the residue extracted with alcohol, which will remove many impurities, but will not remove any of the sulphate of potassium; this residue should be dissolved again in distilled water, and the solution tested for both sulphuric acid and potassium.

XXVIII. Hypochlorite of lime.

368. Bleaching powders.— Bleaching powders are usually prepared by a mixture of hypochlorite of lime with calcic chlorid, and sometimes with a little caustic lime; to this is added hydrochloric acid. Another method is the addition of hydrochloric acid to manganese dioxid, and sometimes the addition of sulphuric acid and common table salt. The action of these forms of bleaching powder will produce poisonous effects, from the free acid and the caustic lime, which will be mainly upon the primæ viæ. In the manufacture of these bleaching powders, free chlorin gas would be evolved, and the accidents from inhalation of this gas would occur from the local action of chlorin. This is described under the head of chlorin and hydrochloric acid. The hypochlorites are decomposed by the carbonic acid (carbon dioxid) in the air.

CHAPTER IV.

ORGANIC POISONS.

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I. ANILIN AND ITS DERIVATIVES.

369. Occurrence.—Anilin is largely used in the arts as a basis for the so-called "anilin dyes." Many of its derivatives form colored substances, which are extensively used in dyeing. Most of the cases of anilin poisoning which are of forensic interest are accidental, though a few have been suicidal.

370. Anilin dyes in general.—Many cases of accidental poisoning, some of them severe, have occurred from the wearing of articles of apparel dyed with anilin dyes. In many cases the poisoning has been directly attributable to anilin oil. In others, it is a question whether the anilin dye was the cause of the symptoms, or whether the symptoms were not due to some impurity left in the dye in its preparation, or in the process of dyeing. In many cases the symptoms were those

of arsenical poison. Chief among the impurities may be mentioned arsenic; other impurities are lead, sulphuric acid, carbolic acid, and various mordants. Some of the salts of anilin or allied hydrocarbons are apparently more poisonous than the base itself. This may be due to their greater solubility as a rule, or that they are more easily acted upon by the digestive juices.

371. Properties.—Anilin, amidobenzol, $C_6H_5NH_2$, is a colorless, inflammable oil, with a peculiar odor and aromatic taste. It is slightly soluble in water, readily so in alcohol and ether. It forms a series of well-defined salts with acid radicles. Boiling point 184°.

372. Derivatives of anilin.— Acetanilid, $C_6H_5NHCOCH_3$; phenacetin, $C_6H_4NHCOCH_3.OC_2H_5$; exalgin, $C_6H_5NCH_3COCH_3$; antipyrin, $C_6H_5NCOCHNCHCCH_3$, and many other derivatives of anilin, are largely used in medicine as antipyretics.

373. Properties of anilin derivatives.—As a rule they are white, crystalline bodies, without much odor, soluble in water, alcohol, and chloroform; and the aqueous solution has a bitter taste.

374. Reactions of acetanilid in general.—Solutions of acetanilid with potassium hydrate and chloroform give the disagreeable odor of phenyl isocyanid.

Boil a few centigrams acetanilid with 1 cubic centimeter potassium hydrate, and suspend a drop of filtered, chlorinated lime solution on a glass rod over the hot mixture. The drop is soon colored yellow (with a violet tinge by reflected light); upon continued heating, it turns violet.

Heat a little of the suspected substance for a minute with 1 cubic centimeter hydrochloric acid, and then add a little of a solution of calcium hypochlorite and phenol. A red color is produced, changing to blue on the addition of ammonia.[1]

375. Antipyrin in general.—Ferric chlorid solution produces a red color with antipyrin, which is discharged by mineral acids.[2]

A drop or two of sodium nitrite, acidified with sulphuric acid, added to a little of a solution of antipyrin gives a green color (nitroso-antipyrin). Delicacy 1 : 1000.[3]

376. Phenacetin in general.—Ten to twelve per cent nitric acid, boiled with powdered phenacetin, yields yellow, crystalline needles. Antipyrin and acetanilid do not respond.

[1] Zeitschr. f. anal. Chem. XXVII. p. 666.

[2] Jahresberich, d. Chem. 1886, 1893

[3] Knorr. Liebig's Annalen, CCXXXVIII. p. 212.

Vapors of phenacetin, heated with zinc chlorid, color wood shavings yellow, but residue is reddish yellow (acetanilid yellow).

377. Action of anilin.—Anilin coagulates albumin, and is one of the poisons which act upon the blood by destroying the vitality of the blood corpuscles. This action interferes with its property as an oxygen carrier to the tissues of the body, which is essential to the interchanges of gases (metabolism) in these tissues; in other words, the physical condition of the blood corpuscles undergoes a disintegration.

378. Action of acetanilid.—"Acetanilid," a proprietary article on general sale in commerce, is a derivative from anilin, and has been used to reduce febrile conditions, by its peculiar action of reducing the strength of the circulation, and by its secondary influence upon the heart's action.

379. Other derivatives of anilin and their action.—Exalgin is another derivative from anilin. Other derivatives are phenacetin, lactophenin, malakin, phenocoll, etc. All of these products of anilin probably act upon the body by the development in the tissues of a simpler derivative, through their decomposition in the tissues; some decompose more rapidly than others, and hence, probably have a more intense action because of this more active decomposition. There have been numerous cases of poisoning during the earlier use of the anilin derivatives in medical practice; but, as they were prescribed by educated, medical practitioners, these poisonous symptoms were cared for, without very serious or fatal effects. They have, however, been compounded and sold as "headache" powders or tablets, under proprietary names. Anilin derivatives have also been used as dressing for wounds, since anilin has antiseptic properties; and sometimes, as a result, poisonous symptoms have arisen. Two cases are reported by Massachusetts medical examiners, one of a child (1894) aged one year, who ate "some" of the tablets containing acetanilid, and died. There was no autopsy. Another case of fatal poisoning was also returned by a medical examiner, as occurring in 1899.

380. Symptoms of anilin poisoning.—These are excited, reflex actions with increased sensitiveness of the skin, depression of the pulse rate and strength, with, at first, an elevated body temperature, followed by its decrease. The most marked symptom is cyanosis, in which the color of the skin becomes gray and of a bluish tinge, due to the production of fine, black granules in the blood; the patient has, sometimes, an odor of anilin emanating from surface of body. The separation of the anilin particles, which pass out through the kidneys,

often causes an inflammation of that organ, and produces nephritis; sometimes blood itself is voided with the urine. Various forms of exanthematous eruption may appear on the skin; convulsions, delirium, and coma may supervene.

Brown[4] describes a fatal case in which there were the following symptoms in detail: General hyperesthesia, and increase in the reflex response to external irritation; temperature increased, and afterwards decreased; persistent nausea, vomiting, and delirium; cyanosis was marked. An examination of the richness in oxygen of the blood showed, on the eighth day, that the number of blood corpuscles was 1,116,000 with 30 per cent hemoglobin (normal number per cubic millimeter being 4,000,000 to 5,000,000). This was on the day of her death. The white corpuscles were increased in number (66,450), and there were a large number of nucleated red cells of all sizes. Twenty-four hours after she took the poison, bloody urine was voided, and complete suppression of urine three days before her death. There were acute nephritis, intestinal hemorrhage, and acute and increasing jaundice.

Dr. F. J. Smith reports[5] another case: A woman, forty-two years old, drank about 3 fluidounces of marking ink, the greatest part of which consisted of anilin; in a short time she became unconscious, and remained so until she died, twelve hours after swallowing the ink. Eighty minutes after taking the poison her lips were dark purple; the surface of her skin was white with a bluish tinge; the pupils of the eyes were small, and did not respond to the stimulus of light; her respiration was stertorous; the pulse rate slow (60 per minute), but strong. The stomach was washed out, ether injected, and oxygen administered; the patient, however, died in a state of coma.

381. Subacute symptoms.— As the derivatives of anilin, acetanilid and others, have been frequently employed, very often without the advice of a physician, the effects of this drug, either as a subacute intoxication or in its chronic form, have been observed. These are cyanosis, jaundice, anemia, and asthma. Some individuals are more susceptible to its poisonous action than others, in some forms of disease, especially when, as, for instance, there is an existing anemic condition, or some defect in the functions of the heart, notably in typhoid fever. Persons addicted to oft repeated doses suffer in the interim from headache, loss of muscular power, diminished sensibility of the skin, nausea, decreased appetite, pallor of the complexion,

[4] Am. Jour. of Med. Sci. 1901, p. 770. [5] London Lancet, Jan. 13, 1894.

eczematous eruption on the skin, and a general feeling of malaise. This chronic form of poisoning is more generally seen in the workmen in dye factories, where anilin is manufactured on a large scale.

382. Action of anilin.—Engelhardt[6] has found that anilin black is formed in the blood, probably as the result of oxidation, and fine black granules are observed in every drop of blood; in consequence of this, a refraction occurs, developing a pale or gray-blue tinge of the skin. The effect of an alteration of the physico-chemical condition of the blood corpuscles (oxygen carriers), as observed in the case of other poisons which accomplish the same effect, naturally prevents the interchange of the gases concerned in oxidation of the tissues (metabolism), which is necessary to maintenance of life. This interruption to the oxidation in the tissues would be marked by a disturbance in the functions of the organs of life; for example, in addition to the symptoms noted in our preceding paragraph, the functions connected with the nerve centers, as well as with their peripheral distribution, would be lessened, even aborted, and paralytic symptoms would be manifested; at first, there would be observed an increased reflex action, to be followed by its decrease.

Schmiedberg has detected a complex chemical combination, para-amido-phenol-ether-sulphuric acid, which appears as an alkaline salt in the urine. The urine is usually brownish or of a black-brown color, and may contain hyaline, cylindrical casts, if the life of the patient is prolonged sufficiently to allow the kidney to eliminate the poison through this channel. In the case of rapid death following ingestion of any anilin poison the spectroscope will show the absorption band of methemoglobin. A further illustration of the effect on the nerve center of the deposit of anilin black from the blood is suggested by the appearance of amblyopia in chronic poisoning.

383. Lethal dose.—Mr. Blyth[7] states that "this is not known; but an adult would probably be killed by a single dose of anything over 93 grains (6 gm.). Recovery after treatment has been known after 154 grains (10 gm.); the fatal dose for rabbits is 15 to 23 grains (1 to 1.5 gm.), for dogs 3 to 5 grains (.2 to .32 gm.)." The fatal doses in the two cases related in a previous paragraph have been there stated. (P. 379.)

384. Treatment.—Sodium sulphate should be administered after washing out the stomach to remove unabsorbed poison which has been swallowed. Inhalation of oxygen gas should be used to counteract,

[6] Beiträge zur Tox. des Anilins. Inaug-Disser. Dorpat, 1888.

[7] Op. cit. p. 281.

if not too long delayed after the taking of the anilin derivative, the physico-chemical action on the blood corpuscles; if the vapor of anilin oil has been inhaled in sufficient strength to be absorbed through the lungs into the blood, the inhalation of oxygen is pre-eminently indicated, as well as to remove the patient into a pure and warm air. It is specially important to remove any of the poison from the external surface of the body, in view of the fact that this poison can be absorbed through contact with the skin.

The effects of chronic poisoning should be treated by heart stimulants, but never by alcoholic stimulants, and by a general treatment to counteract the tendency to displace the oxygen in the blood corpuscles. Though there is no mention made in the ordinary text-books, it would seem good practice to combat the anemic condition, consequent to chronic poisoning from the anilins, by a careful use of the milder preparations of iron, for the purpose of improving the defective metabolism met with in persons subjected to the inspiration of anilin oil in dye factories during anilin manufacture.

385. Post-mortem appearances.—The pathology of poisoning from the anilins does not, unless we except the spectroscopic test for methemoglobin in the blood, amount to anything characteristic of its action. The odor of anilin is very often strongly evident as emanating from the body.

386. Detection and tests.—The material to be tested is made strongly alkaline, and then distilled with steam. The anilin is distilled with the steam and is found in the distillate; if in large quantity, it separates as an oily layer. The aqueous distillate containing the anilin is then shaken out with ether, and the ether evaporated, leaving the anilin as a residue.

Anilin with a drop of concentrated sulphuric acid and a small crystal of potassium bichromate gives first a red and then a blue color, which disappears slowly. With fuming nitric acid, anilin gives a deep blue solution, becoming yellow and finally red, upon warming.

A very dilute aqueous solution of anilin, treated with a solution of chlorinated lime, and then a few drops of a very dilute ammonium sulphid solution, develops a rose-red color. Sensitive 1:250,000. Phenol and sodium hypochlorite solution added to an aqueous solution of anilin gives a dark blue color, changed to red by hydrochloric acid. A pine shaving, moistened with a very dilute solution of an anilin salt, is colored yellow. A very dilute solution of an anilin salt yields with bromin water a precipitate of tribromanilin.

The material under examination is first acidified, then treated with

ethyl alcohol in large excess. This coagulates the proteids. The filtrate is evaporated to a small volume and again precipitated by alcohol. This filtrate is concentrated, and, after the removal of all the alcohol, adding water if necessary to keep the solution fairly dilute, it is shaken out with ligroin, then with chloroform. The chloroform will extract most of these substances. The residue, after evaporation of the chloroform, can then be tested for the suspected substance, by special tests in each case.

II. Acetic acid.

387. **In general.**— Acetic acid, $CH_3\ CO.OH$, is the acid of vinegar. It is manufactured for commercial purposes by the destructive distillation of wood. The pure acid, glacial acetic acid, is a colorless liquid at ordinary temperatures, with a strong, pungent odor and bitter taste. It crystallizes at 17°, and boils at 119° Soluble in water, alcohol, and ether, in all proportions.

Deaths from this acid have been most generally through its being swallowed in the form of glacial acetic acid by accident. Dr. Luff records one case in which there were suspicions of attempt to murder. In this case (*Reg.* v. *Newber,* C. C. C. 1894), the accused servant girl was convicted of murdering a child of ten months, with about a teaspoonful of the glacial acid, by pouring it into the mouth. Mr. Blyth records in his ten years' table, previously referred to, nine deaths from poisoning by acetic acid as occurring in England and Wales. Dr. Luff records a second case of a boy, four and a half years of age, who died the following day after drinking an unknown quantity of the glacial acid. This was a death by accident. In addition to the above eleven cases of fatal poisoning from the strong, glacial acetic acid, we find reported but seven cases of poisoning by it, one by Orfila,[8] one by Taylor,[9] and two by Heine and Hergott.[10] In Orfila's case the liquid was swallowed by a young girl, and in a few hours afterward she died in great agony.

In Taylor's case, a woman gave by mistake to her child of two years a dose of glacial acetic acid, which had been used for removing warts; the child suffered the most intense pain, and died in about thirty-six hours. In the two fatal cases of Heine and Hergott an injection of Villati's solution (sulphate of copper 4 parts, sulphate of zinc 4 parts, solution of subacetate of lead 7 parts, acetic acid 52

[8] Toxicologie, Vol. II. p. 198.
[9] On Poisons, Phila. 1875, p. 241.
[10] Ziemssen's Cyclopedia of Medicine, New York, 1878, p. 349.

parts) was used in a suppurating wound. The remaining three cases were attempted suicides, or where the acid had been taken by mistake for a medicine, only one of which ended fatally.[11] In Heine's case the injection was immediately followed by hemorrhage from the wound and violent local pains in it; these were followed by prostration with pallor of the skin, trembling of the entire body, accompanied with chill, and a rapid and weak pulse, the patient dying in a state of collapse in a few hours. In Hergott's case nausea and vomiting occurred, and death in a very short time.

Officinal acetic acid (U. S. P.) contains 36 per cent of glacial acetic acid, and of course would be followed by similar symptoms as those accompanying the ingestion of the pure acid, *viz.*, irritant and corrosive local action, with symptoms of collapse, muscular twitchings, and chills.

Dilute acetic acid (U. S. P.) is made by adding to 100 parts of the officinal acetic acid above mentioned 500 parts of water (distilled) and this can be taken in apparently large quantities without danger to life, though many standard books speak of a chronic poisoning from the daily use of vinegar or weak acid by hysterical women, for the purpose of blanching the complexion. It is more probable that anemic persons, who practise this injurious habit, crave an acid to relieve their restlessness and vitiated appetite, and thus prevent the natural desire for good and wholesome food. This would result in increasing an already depraved process of nutrition, and impair still further the digestion. Hence the use of vinegar or weak, diluted acetic acid is not the prime factor in inducing the gradual exhaustion, and the consequent deterioration of health.

Dr. David, of Montreal, met with an instance in which a woman endeavored to destroy herself by drinking a quantity of common vinegar. When seen, three hours afterwards, her countenance was wild and the pupils dilated, the body was covered with a cold perspiration, and the breathing was laborious and hurried. Her tongue was dry and cold, the abdomen distended, and she had acute pain in the epigastrium, which was increased by pressure. She was, moreover, delirious. She soon recovered after the administration of an emetic of sulphate of zinc.[12-3]

388. Symptoms of poisoning.—The acute symptoms of poisoning by glacial acetic acid are, as in the case of the mineral acids, a corrosive

[11] Husemann, Toxicologie, Birkett, Lancet, 1867, Mèliou, Frank's Magaz. in Ziemssem's Cyclopedia, op. cit.

[12-3] Am. Jour. of Med. Sci. Oct. 1848, p. 302.

local action upon the inner surface of the mouth, throat, and the alimentary tract, and also upon the fauces and entrance of the air passage. The effect of this corrosion will produce pain at the epigastrium, vomiting of a liquid smelling strongly of the acid; to this will ensue a difficult respiration, and coughing up of large quantities of mucus; on the second and third days a mucopurulent matter will be expectorated, and, perhaps, marked signs of a localized pneumonia, which, if the patient is to recover, will clear up in the course of a week or ten days. If a fatal dose has been swallowed, the severe effects of corrosion of the lining membrane of the gullet and stomach, collapse, and convulsions will follow; if the acid has been drawn into the larynx and trachea, the difficulty of breathing from edema of the glottis and primary air passages may be so serious as to prevent the entrance of air into the lungs, and so death may rapidly ensue from asphyxia. Death may thus result in a very short time and within the first twenty-four hours.

In the subacute form of poisoning incident upon the swallowing of the glacial acetic acid, we may see a continuance of the vomiting, which may consist of portions of the mucous membrane which lines the œsophagus and stomach; we may also find a secondary inflammation of the air passages, which, as above noted, may induce a traumatic pneumonia.

In the chronic form of poisoning we may have to deal with the result of the local action upon the mucous membrane of the primæ viæ, of the alimentary tract, and of the air passages, as well as that of the lungs.

389. **Diagnosis.**—The pungent smell of acetic acid will mark the presence of this substance, in exhalations from the throat, as also from the warm, vomited matters from the stomach; ordinarily, the lips and anterior part of the mouth escape the most serious, corrosive action on the mucous surface, while the white, softening, local aspect of the back of the tongue, fauces, tonsils, and larynx, marks the caustic effects of this acid.

390. **Lethal dose.**—In the two cases mentioned by Dr. Luff, a teaspoonful of the glacial acid was given to an infant of ten months, which caused death, but the amount taken by the boy of four and a half years was not known; in his third case, a woman twenty-one years of age, the amount swallowed was about two teaspoonfuls, from which she recovered with treatment of a mixture of magnesia and water in large amount.

391. **Treatment.**—Magnesia should be administered in a large

amount of water to counteract caustic action on the mucous membrane of the mouth, gullet, stomach, and intestinal canal, and as gargle for the throat; if the edematous swelling of the larynx and trachea obstructs the inspiration of air into the lungs, tracheotomy will be indicated. It should always be remembered that acetic acid is volatile, and that its vapor can thus be inspired, though the liquid itself may not pass into the air passages.

392. Post-mortem appearances.—In addition to the local, whitened, caustic action on the mucous membrane of the mouth and fauces, there have been observed patches of erosions of the mouth, epiglottis, larynx, and œsophagus, as also of the stomach; the mucosa of the stomach may also be corrugated, and show patches of injection of the submucous tissue.

393. Detection and tests.—In case of an investigation to detect this acid in the contents of the stomach, it must be remembered that it is contained in the natural secretions of this organ; hence, to be of any value, the chemical evidence must detect a considerable quantity of it. Also, as Christison suggests, vinegar is a common remedy with the vulgar for many diseases, and especially for poisoning.

The material under investigation is best distilled, after the addition of tartaric acid, in a stream of carbonic dioxid. The acetic acid is found in the distillate, and its presence confirmed by special tests.

Warmed with sulphuric acid it blackens; with $AgNO_3$ a white, crystalline precipitate, partly dissolved by heat, but giving no reduction of silver on boiling. Neutral solution of ferric chlorid produces, in neutral solutions of acetates, a deep red color, and, on boiling, a precipitate of a basic acetate of iron.

III. Carbolic acid.

394. In general.—Carbolic acid has, of late years, given rise to quite a number of cases of poisoning, chiefly suicidal and accidental. It is obtained from coal tar, and, when perfectly pure, is seen in the form of white, colorless, needle-shaped crystals, which, after standing for a long time, have a pinkish color when seen in bulk. It absorbs moisture readily, and becomes converted into an oily liquid, having a reddish or brownish color. It has a penetrating, unpleasant odor like that of creasote. It is soluble in 20 parts of water, and is very soluble in alcohol, ether, and chloroform.

It deliquesces to form a hydrate, which crystallizes in prisms known as "ninety-five per cent acid." The crystals of the pure

acid melt at about 40°, those of the hydrate at 16° C. The crude carbolic acid of commerce is a reddish liquid used as a disinfectant.

Carbolic acid, so easily accessible to the general public, and simulating, both as regards taste and smell, common whisky, has been recognized as a poison since the first fatal result of its being taken to produce death in 1864. Mr. Blyth gives, in his ten years' mortality by poisons, the total of 373 deaths from this substance. In the mortality table of the Massachusetts medical examiners, contained in our first chapter (pp. 18, 20), 176 deaths by carbolic acid are reported during the twenty-five years covered in that table. Dr. Luff states that "a parliamentary return of the number of deaths in England and Wales, caused by the taking of carbolic acid, shows that in the five years from 1887 to 1891 there were 375 deaths from this poison. These were by accident, 138; suicide, 236; murder or manslaughter, 1; of the cases of suicide, the greater number occurred among females."

It should be borne in mind, as is justly remarked in Mr. Blyth's book on poisons before referred to, "that commonly the word 'carbolic acid' is not used by coroners, juries, or medical men, in a strictly chemical sense, the term being made to include disinfecting fluids, which are almost wholly composed of the cresols and scarcely any phenol."

He also states that "at present (1895) it occupies the third place in fatality of all poisons in England." There were, during the ten years preceding the publication of his book, 741 people killed by carbolic acid, though only 1 by murder; the other deaths from it were either accidental or by suicide. He mentions 4 cases in which carbolic acid was used as a rectal injection to dislodge intestinal worms; 1 of these cases ended fatally, the other 3 recovered.

There are two cases of suicide reported in the medical examiners' returns to the secretary of state of Massachusetts, in both of which a disinfectant under the name of "cresoline" was swallowed; one, in the year 1899 by a dose of 3 ounces (89 c.c); and the second in 1900, of several ounces. This mixture is supposed to be made of a number of constituents, among which is cresol (cresylic acid, methyl-phenol), and is of an alkaline character. Cresol has the formula chemically represented: $C_6H_4.OHCH_3$.

395. Symptoms of acute poisoning.—In the early days, when the antiseptic treatment in surgery was in vogue, topical applications of carbolic acid to the skin or wounds would be followed by disturbance of the digestion, loss of appetite, nausea, dizziness, ringing in

the ears, headache, etc.; sometimes, also, inflammatory action would be observed in the kidneys; in some of these cases convulsion would appear and be followed by symptoms of narcosis, such as mild delirium, irregular respiration, and sleepiness. The nephritis (inflammation of kidneys) would be marked by the presence of albumin in the urine, blood, etc., and the urine itself would have a dark color, increasing on exposure to the air.

When swallowed, the strong acid will almost immediately, as in the case of the corrosive acids, be followed by a burning pain in the primæ viæ, and the mucous membrane of the lips, mouth, and throat, will become white; stains will also be observed upon the skin outside of the mouth. The pungent odor of carbolic acid is very pronounced. Vomiting is not always present, because the anæsthetic effect, numbness, upon the mucous surface of the alimentary tract is a peculiarity of this poison. Difficult respiratory efforts appear, which become stertorous. The pupils of the eyes do not respond to light and are contracted; the mucous surface of the eye does not respond to irritation of touch, having lost its reflex irritability. Unsteadiness of gait, collapse, sometimes convulsions, and, generally, coma precede death within one to twelve hours. When applied to the skin, carbolic acid produces caustic and benumbing effect. The extensive use of this acid as a disinfectant and for other purposes renders it a common article of household use, and explains the numerous, recent cases of poisoning by it. Falck[14] has collected since 1868, from medical literature, 85 cases of poisoning by it, of which 7 were suicidal, 39 were due to its use as a surgical dressing or wash, and 39 were cases of accidental poisoning, the carbolic acid having been taken by mistake for some medicine or drink. Most of the cases of acute poisoning by carbolic acid are due to the ingestion of crude carbolic acid, which is usually a brown, oily fluid, having the odor and properties of carbolic acid, although it is impure. The poisonous action of strong carbolic acid is that of a caustic and corrosive, upon the tissues of the body; upon the skin it produces a white streak; upon the mucous membranes an edematous, swollen, and inflamed condition, and a bleached appearance, the mucous membrane often being readily peeled off from the subjacent tissues. The symptoms which follow the swallowing of a poisonous dose are twofold, depending very much upon the degree of concentration of the acid. Calvert's No. 1 acid is crystalline at a temperature of 60° F., or 15° C., and is a strong, local

[14] Lehrbuch der prakt. Toxicologie, 1880, p. 208.

caustic, benumbing sensibility to pain soon after the first moment of contact. The ingestion, therefore, of this form of the acid is followed immediately, or in a very few minutes, by narcotism and unconsciousness, relaxed or flaccid condition of the muscular system; the pupils are insensible to the stimulating action of light, sometimes of natural size, but more often very much contracted. The face is livid, and, on the skin near the angles of the mouth and running down to the chin, white streaks may be observed, showing the local, caustic action of the drug. The breathing is labored and slow, pulse is small and slow, oftentimes regular, but most generally the pulse beats are irregular and intermittent in character; in about an hour after reception of the poison, the intermittence and irregularity of respiratory movements and pulse beats are very peculiar; for a few seconds they may be slow (about 40 to the minute for the pulse with about 9 respirations), then both may be rapid, the pulse 130, and the respirations 45 to the minute. These peculiarities in pulse rate and respiratory movements are rather peculiar to the poisonous action of carbolic acid, and are also evidences of its marked, narcotic action. The heart sounds are pure, though the pulsations are usually tumultuous from weakness of the cardiac muscular fibers. These peculiarities of respiration and pulse become more marked as death approaches, and resemble the dying moments of a patient with fatal cerebral disease.

The other prominent symptoms, which would usually be observed in persons who have taken a less concentrated form of the acid, resemble those observed with any other corrosive; these are pain in the mouth, gullet, and stomach, which is soon followed by insensibility, with difficult or impossible deglutition, cold and clammy skin, decreased surface temperature (below 97° F., 37° C.), stertorous respiration, a small, frequent, and intermittent pulse, contracted pupils, insensibility to pain, and the voiding of a brownish, blackish, or greenish urine. The nervous system is profoundly impressed. The local action upon the intestinal canal is that of a corrosive or irritant poison, causing usually a white eschar, the skin and mucous membrane becoming cornified.

A case reported by Mr. Silk,[15] resident in the University College Hospital, London, is that of a person who had taken about a wine-glassful of strong carbolic acid an hour before his reception into that hospital. Coma and stertorous breathing were quite well marked. Eyeballs were prominent, and the pupils contracted to a pin's point and insensible to the action of light or touch. Lips were shriveled,

[15] Brit. Med. Jour. April, 1881 p. 640.

and the skin was white about the angles of the mouth, with excoriations. Pulse full and soft, occasionally intermittent, 120 per minute. Temperature of the body was under 95° F., 35° C. Surface of the body cold, clammy, and apparently without sensation. Death occurred in about three and a half hours after swallowing the acid. A case of poisoning, in a woman forty-five years of age, was brought to the Charing Cross Hospital, London, of which Mr. Harrison presented the following notes.[16] He saw the patient half an hour after she had taken the acid. The friends reported that no pain or delirium had been observed; but that, immediately after the ingestion, insensibility appeared. On entrance to the hospital, breathing was stertorous, pupils were insensible to light or touch, and were somewhat contracted. The smell of carbolic acid evolved from her person was very pungent, and the skin about the mouth was reddened; the pulse was small and irregular, and the surface of the body was cold. The stomach pump removed about half a pint of dark, grumous fluid, smelling strongly of the acid. The patient died in about three quarters of an hour from the time of swallowing the poison.

Dr. Kirk reports in great detail[17] the symptoms of a fatal poisoning of a woman; she took an unknown amount of Calvert's No. 1 acid (less than 2 ounces). Narcotic symptoms and unconsciousness came on in a very few minutes. She was seen by Dr. Kirk in less than half an hour after taking the poison. "Her whole body was flaccid, her pupils insensible to light, and were neither increased nor diminished in size. Her face was livid, and from the corner of the mouth a white streak passed downwards to the chin. Her breathing was slightly labored; the respirations 8 per minute. Pulse 40, small, but regular. Her extremities were cold. At six o'clock, or fifty minutes after the poison had been taken, her respiration became irregular." From this time onward until her death, four hours after swallowing the drug, the respiratory movements and pulse beats showed alternations of increased and diminished rates of no particular regularity of intervals. At one time the pulse beat regularly for seventy seconds, about 40 per minute, with 9 inspirations per minute, and then the pulse movements were 100 per minute, respirations 30 per minute, shallow and gasping. Again, about one and a half hours after the acid was taken: "The pulse was beating about 50 per minute, and, after fifty seconds, it became rapidly quicker until it reached 130, at which rate it continued for half a minute, and then fell as

[16] Med. Times and Gazette, Aug. 30, 1879.

[17] Glasgow Med. Jour. June, 1881, p. 433.

quickly as it had risen. The respirations during the pause between the accelerations were not more than 8 per minute, deep and quiet. Almost simultaneously with the pulse, the rate of respiration increased in twenty to twenty-five seconds to 45 per minute, and then fell with the pulse." The behavior of both respiratory and heart movements was very peculiar and intermittent. "The heart sounds were quite pure, but the beating was tumultuous, and the contractions appeared to be only partial. The respirations now were very slow, about 2 to the minute, very deep, not stertorous." In two hours and a half from the time the poison was taken, "the time between the acceleration of the pulse and respiratory rates increased, the pulse became slower, and the respiration ceased for a longer or shorter period" until three and a quarter hours had passed, when the pulse beats were only 20 or 30 per minute and very feeble, and then became so accelerated that the intervals between each pulsation could hardly be distinguished, the respiration partaking of the same general character as is often witnessed at the dying moments of a patient with fatal cerebral disease.

Dr. Reichert[18] reviews the symptoms of poisoning in fifty-four cases collected by him. The most rapidly fatal case in this synopsis occurred in three minutes after a dose of ½ ounce (15.55 gm.) of impure acid.[19] Of the other cases, one was reported[20] where death occurred in ten or fifteen minutes, and another of a child[21] who had taken by mistake a tablespoonful of carbolic acid, followed by copious draughts of warm water, and who died in seventeen minutes. The longest duration of life after a fatal dose of poisoning by this acid was two days.[22] The smallest fatal dose recorded in this class of cases was 1 dram (3.9 gm.) taken by a man sixty years of age; but two children, who took a similar dose recovered.

A case is recorded in 1902 of a suicide who swallowed an unknown quantity of carbolic acid, which was followed by death within ten minutes. Another case is recorded, wherein death followed the ingestion of 1 ounce of carbolic acid in crystals, within fifteen minutes (1892). The ingestion of 2 ounces of the pure acid was followed by death in less than thirty minutes (1895). In these records for the year 1898 there are three cases reported, dose not stated, where death occurred, respectively, in three hours, in four hours, and in five

[18] Am. Jour. of Med. Sci. Oct. 1880.

[19] See the Phila. Med. Times, 1873. Vol II. p. 284.

[20] Hill. Richmond and Louisville Jour. and N. Y. Med. Rec. 1873.

[21] Louisville Med. News, 1880, No. 10, p. 294.

[22] Med. Times and Gazette, 1870, p. 474.

hours, after swallowing carbolic acid. A case is reported in 1895, where, after death, the odor of carbolic acid was apparent when pressure was applied over the epigastrium. In another case of poisoning after swallowing a dose of carbolic acid, the dose of which was assumed, from what information could be obtained, to be 3 drams, death occurred within forty minutes. The following note of the autopsy in this case is reported: Stomach contained about 4 ounces of undigested food, smelling strongly of carbolic acid; its tissue was engorged, mucosa destroyed, corrugated, and shriveled. Liver, kidneys, and urine all smelled strongly of carbolic acid.

396. Vapor of carbolic acid.—Death from poisoning by this acid can also occur apparently from simple inhalation; for Dr. F. P. Blake[23] reports the case of a woman suffering from neuralgia, who sought for relief from her pain by inhaling the acid. She was found dead in her bed with a handkerchief inwrapping a sponge and applied close to her face, which was livid. There was a strong smell of carbolic acid in the room, and the sponge and handkerchief, though dry, were strongly impregnated with its odor; the appearance of the inside of the mouth, the surface of which was free from excoriation or caustic marks, did not indicate that she had taken any of the acid in her mouth, and the tip of the nose was blistered from contact with the cloth and sponge.

397. Poisoning from external use.—Dr. Paul[24] reports a case where constitutional symptoms of poisoning occurred from the external application of carbolic acid over a surface of about 5 square inches, of which only a small portion was denuded of cuticle. The symptoms began twenty minutes after the application. In this connection it must be said that susceptibility to carbolic acid intoxication absorbed by the lungs or from the skin is very variable, and in some persons occurs on very slight provocation. Proofs of intoxication are shown by the peculiar appearance of the urine, before alluded to. Danger of criminal poisoning by carbolic acid is not great, since its strong and pungent odor would make it difficult to administer without the knowledge of the victim; hence, when not the result of accident, a death from this agent might reasonably be assumed to be one of suicidal, not homicidal, intent. It must be said, however, that the smell and brown color of the strong solution of the acid may be mistaken for smoky whisky, by persons in whom the sense of smell is not delicate. In fact, most cases of poisoning by this agent, which have been recorded, are either those of carelessness or suicide.

[23] Phila. Med. Times, July, 1881. [24] *Ibid.* May 8, 1880.

Cases of death from surgical dressings with carbolic acid have been reported. One,[25] reported by Dr. Bradford, is not conclusively a death from carbolic acid intoxication, though it is evident that a large amount of this agent was absorbed from the inflamed tissues in a cold abscess of the hip, which was washed out and distended by a 2½ per cent solution of carbolic acid, and dressed antiseptically with carbolized gauze. In this case the lesions of greatest importance were in the heart and kidneys, which were the seat of fatty degeneration, and they were analogous to the appearances of poisoning by phosphorus, arsenic, or ammonia. Dr. Bradford says, "The clinical history of the case directly suggests that the lesions found may be attributed to the toxic action of carbolic acid." The urine, passed within the first twenty-four hours after the carbolic acid was used, "was tar color," and its chemical analysis showed a "color slightly smoky and the presence of a very large amount of a sulpho-acid (presumably sulphophenylic)." In an article by Mr. Macphail,[26] the subject of poisoning by absorption of carbolic acid dressing is treated at length; this depends upon the fact that the solution of carbolic acid is in contact with tissues or serous membrane, inflamed, and abnormally active in absorbing deleterious substances. It is well known in surgery that decomposing fluids, pus, and fetid discharges may be readily absorbed by contact with diseased surfaces, and thus give rise to septicemia or blood poisoning. It is reasonable, therefore, to explain the absorption of carbolic acid upon the same principle. In fact, Professor Lister, the earliest advocate for antiseptic dressings,[27] says, "I believe the secret of our immunity from these (carbolic acid) toxic effects is that we avoid as much as possible all unnecessary action of the carbolic acid upon the tissues. Had I in this boy's case (a case of pleuritic abscess) injected carbolic acid lotion into the pleural cavity, and done this at every dressing . . . I think it is in the highest degree probable that he might have suffered from carbolic acid poisoning. . . . I cannot point to one single instance in which I can be sure that we have had carbolic acid poisoning of any moment whatever, either in my hospital or private practice, during the two years I have been in London."

398. **Its injection into cavities.**—Nusbaum, of Munich, in his work on antiseptics,[28] mentions a case of carbolic acid poisoning from the forcible and daily injection of carbolic acid into an abscess cavity.

[25] Boston Med. and Surg. Jour. April 7, 1881, p. 324.

[26] Edin. Med. Jour. 1881, 2, XXVII. p. 132–144.

[27] Lancet, Dec. 20, 1879.

[28] Leitfaden zur antiseptischen Wundebehandlung, p. 141.

Dr. Whiteford, of Greenock, England,[29] observed symptoms of carbolic acid poisoning from the external application of a 2½ per cent solution to a ring-worm of the scalp in two children; and here it should be observed that children are especially susceptible to the action of carbolic acid. Three other cases of carbolic acid intoxication are reported by Mr. Macphail,[30] from the use of these dressings, but which finally recovered after their discontinuance. Mr. A. P. Gould[31] reports a case of rapid death, after section of the tibia, by the antiseptic method with carbolic acid. The presence of septicemia was negatived by the absence of any sufficient source of septic absorption and of fever. The symptoms observed were similar to those reported by Dr. Bradford,[32] *viz.*, vomiting, diarrhea, nervous prostration, and lowered temperature. The patient died within thirty-seven hours after the operation.

Küster[33] reports a case in which a puerperal uterus was being washed out with a 2 per cent solution of carbolic acid on the fourth day after delivery, without any serious results; but on the next day the use of a 5 per cent solution was followed by the severest symptoms of poisoning, and death on the ninth day.

This form of poisoning by the use of carbolic acid as a surgical dressing or wash can always be prevented by careful examination of the urine. When the acid is absorbed, it pairs with the sulphuric acid radicle to form phenylsulphuric acid, which has no deleterious action upon the economy, so that, as long as it can find any sulphuric acid to unite with, we see no toxic symptoms in the patient, and this event is characterized by the entire disappearance of sulphates from the urine. Ordinarily, if we add to urine acetic acid and a solution of chlorid of barium, an abundant precipitate of sulphate of barium will be formed, but if all of the sulphates have been decomposed by carbolic acid to form the sulphophenylic acid,[33a] no such precipitate will occur; if, at this time, alkaline sulphates, such as the sulphate of sodium, which is found in many mineral waters, be given to the patient, it will neutralize the excess of carbolic acid and prevent toxic symptoms. Baumann[34] reports six cases in support of this action of the sulphate of sodium. In this connection it is important to remember that carbolic acid is entirely eliminated from the system in from twelve to sixteen hours.[35] The amount of carbolic acid which is daily

[29] Related by Mr. Macphail, op. cit.
[30] Op. cit.
[31] Brit. Med. Jour. May 28, 1881.
[32] Op. cit.
[33] Centralblatt f. Gynaekologie, II. 14, 1878.
[33a] See § 403.
[34] Archiv. f. d. ges. Physiog. XIII. p. 285, 1875.
[35] Binnendijk, Jour. de Pharm. et de Chim. 1880, tome 30, p. 515.

eliminated with the urine of a healthy adult, upon a mixed diet, is, according to Engel,[36] about 0.015 gram (about ¼ gr.). The above cases and many others make it evident that carbolic acid in solution can readily be absorbed by inflamed tissues,—especially the serous membranes,—as well as the mucous membranes. When the patient is constantly under observation by a medical attendant, as would naturally be the case after surgical operation, proofs of carbolic acid absorption would most generally be recognized before danger had arisen to the life of the patient. In cases of operations for criminal abortion, the use of carbolic acid, by its absorption, might seriously compromise the life of the patient, and, if performed by an irresponsible or uneducated person, might go forward to a dangerous, if not fatal, issue, without being recognized as a cause. Here, again, it must be observed that the susceptibility to poisoning by carbolic acid in some individuals is much greater than in others, and this fact explains apparent inconsistencies in the reported experience of various physicians and surgeons.

399. Its application over healthy skin.—In Massachusetts Reports, Vol. 138 (1885), pp. 165 *et seq.*, is detailed the trial of a practitioner of medicine, who was convicted of manslaughter. He prescribed that his patient should be wrapped in cloths which were to be saturated with kerosene oil in the following manner: A flannel shirt, flannel drawers, and stockings were to be saturated with the oil, to be placed on her body, and to have the saturation renewed with more kerosene every three hours. This was done with her consent, by her husband and attendants, but not by the defendant personally. After the defendant had gone, in about two hours, owing to the pain and distress of the patient and upon her demand, the saturated flannels were removed, and the defendant was sent for. The husband testified, "On his arrival I told the defendant how my wife had suffered and what we had done; she said it was as if she was in the fire. He replied that it was doing just what he wanted, like a poultice on a boil, drawing it out; that it was her only salvation." Then, being told that the application would not hurt her so much the next time, the flannels were again saturated and replaced. The patient then went to sleep, apparently not suffering as before. The flannels wet with kerosene were continuously applied for two more days, when they were removed by the physician's direction. The evidence showed that the results of this external application caused the effect of blistering and

[36] Ann. de Chimie et de Physique, 1880, tome, 20, p. 230.

burning a large part of the body, removing the scarfskin over two thirds of the surface of the body, taking off the skin over one third of the body, and producing suppurating sores. This treatment was begun on December 29, 1882, and the defendant continued his attendance until January 7, 1883. On that day he was discharged. Other physicians, who were then called in, pronounced her past recovery. She died on January 16th, 1883.

The associate justice of the supreme court (Holmes, at the present time of writing an associate justice of the United States Supreme Court) wrote the opinion of the supreme judicial court in the case. The trial judge ruled, and gave instructions to the jury as follows: "Injurious acts, resulting from gross carelessness or foolhardy presumption, without intent to injure, may constitute an assault. If persons who are engaged in operating steam engines are guilty of gross carelessness or foolhardy presumption, and injuries result, they are criminally liable. So, with apothecaries, if a person, without knowledge and skill, deals with deadly drugs, he may be guilty of gross carelessness amounting to presumption, and be criminally liable. . . . A man is not to be convicted of manslaughter merely because of his ignorance. . . . The defendant in this case is to be tried by no other or higher standard of skill or learning than that which he necessarily assumed in treating her; that is, that he was able to do so, without gross recklessness or foolhardy presumption in undertaking it. It is not necessary to show an evil intent; if, by gross and reckless negligence, he caused the death, he is guilty of culpable homicide."

In affirming a conviction of the accused Mr. Justice Holmes said: "We cannot recognize a privilege to do acts manifestly endangering human life, on the ground of good intentions alone."

The syllabus of the case summarizes the law of the subject in the following words: "If a person, publicly practising as a physician, on being called upon to attend a sick woman, prescribed, with fool hardy presumption or gross recklessness, a course of treatment which causes her death, he may be found guilty of manslaughter, although he acted with her consent, and with no evil intent. An indictment for manslaughter, by causing the clothes of the person killed to be saturated with kerosene, need not allege that the accused knew of the deadly tendency of the kerosene, or that it was of a dangerous tendency."

We make no apology for presenting this legal side of the above case, because the practical law points, as laid down, may serve as a lesson to every practitioner of medicine. This case also serves as

an example of the danger of this poison by its action on the cutaneous covering of the body. By the destruction of the integrity of the skin, and by the absorption of the phenol contained in the kerosene oil, there occurred in the above case anesthesia, narcosis, coma, and death.

Mr. Justice Holmes in giving his opinion that "a man may commit murder or manslaughter by doing otherwise lawful acts recklessly, as, that he may by doing acts unlawful for independent reasons, from which death accidentally ensues," quotes the following authorities: "3 Inst. 57; 1 Hale P. C. 472–477; 1 Hawk. P. C. chap. 29, §§ 3, 4, 12, chap. 31, §§ 4–6; Foster, 262, 263, Homicide, chap. 1, § 4; 4 Bl. Com. 192, 197; 1 East P. C. 260 *et seq.; Hull's Case,* Kelyng, 40, and cases cited below."

The following fatal case is on the returns of the medical examiners to the secretary of state of Massachusetts, for 1889: Reported by Medical Examiner Adams, of Framingham, "a child of two years, June 9th, in Natick, spilled a bottle of strong carbolic acid into a chair, and sat down in it at about 8 A. M. June 8th. A doctor was called, who found the child unconscious at 1 P. M. He died quietly, without pain or convulsions, did not suffocate, but sank gradually away. The whole of the bottom, of posterior surface of legs and of back, was excoriated as high as the region over the kidneys. There were no marks of the poison upon the lips or face. Diagnosis: Poisoning by carbolic acid, chiefly, if not entirely, by absorption through the cutaneous surface."

Among other cases, where the external use of carbolic acid (phenol by some writers) has been followed by serious constitutional symptoms which may end fatally, is one of a newborn child, one week old, who was touched by the fingers of a nurse, whose hands had come in contact with pure carbolic acid; her thumb and one finger simply were placed in contact with the groin of the infant, who died within ten hours. Though the topical action of this acid upon the sound skin is often followed by grave consequences, the dressing on a diseased skin or on denuded surface, composed even of a weak solution of carbolic acid, may provoke poisonous symptoms. This case is mentioned by Mr. Blyth: "A surgeon prescribed for two joiners, who suffered from scabies, a lotion which was intended to contain 30 grams of carbolic acid in 240 cubic centimeters of water; but the actual contents of the flask were estimated afterwards from analysis by Hoppe-Seyler to be 33.26 grams, and the quantity used by each to be equal to 13.37 grams (206 gr.) of carbolic acid. One of the

men died; the survivor described his own symptoms as follows: He and his companion stood in front of the fire, and rubbed the lotion in; he rubbed it into his legs, breast, and the front part of his body; the other parts were mutually rubbed. Whilst rubbing his right arm, and drying it before the fire, he felt a burning sensation, a tightness and giddiness, and mentioned his sensations to his companion, who laughed. This condition lasted from five to ten minutes; but he did not remember whether his companion complained of anything, nor did he know what became of him, nor how he himself came to be in bed. He was holding on to the joiner's bench, looking with wide-staring eyes like a drunken man, and was delirious for half an hour. The following night he slept uneasily, and complained of headache and burning of the skin. The pulse was 68, the appearance of the urine, appetite, and sense of taste were normal; the bowels confined. He soon recovered. The other joiner seems to have died as if he had taken prussic acid. He called to his mother, "Ich habe einen Rausch," and died with livid face, after taking two deep, short inspirations.

Two other cases are related by the same author. In the first, a 2 per cent solution of carbolic acid was applied on a compress to an erysipelatous inflammation on a chicken pox pustule, and the following morning poisonous symptoms appeared, passing of dark-colored urine, sweats, vomiting, contracted pupils, spasmodic twitchings of the eyelids, with strabismus, slow inspiration, and inability to swallow; the child, seven weeks old, died within twenty-four hours. The other case, taken from the American Journal of Pharmacy occurred in this country and related to a boy who had been bitten by a dog; he died within twenty-four hours after a lotion, consisting of 9 parts of this acid to 1 part of glycerin, had been applied to the wound.

These cases of the fatal effects from the topical application of carbolic acid to the skin and other tissues of the body might be multiplied by further citations from other well-recognized authorities on toxicology; but what has been rehearsed is, in our opinion, sufficient to show the great danger of this form of administration.

400. Subacute and chronic poisoning.—From what we have described (see §§ 397, 398) as to the physico-chemical action on the animal tissues, and the conversion of carbolic acid, when absorbed into the animal system, into phenylsulphuric acid, which has no deleterious action upon the economy, we should not expect to find any fatal results to occur after the ingestion of small doses continuously administered, unless fatal result should be produced from one single

dose, as in acute poisoning. Indeed, Falck's careful analysis of 87 cases of carbolic acid poisoning indicates that 71 per cent of these died within twelve hours, and about 91 per cent within twenty-four hours.

401. Lethal dose.—The fatal dose for infants is smaller in proportion to their weight of body than for adults. There is, also, a peculiar susceptibility, in some adults, to the poisonous action of carbolic acid. Setting these conditions aside, we may state on the authority of Falck that the minimum dose for man is 15 grams (231.5 gr.), or about two decigrams per kilogram of body weight. According to his analysis of fatal cases collected by him, in 5 cases 30 grams caused death in five minutes to an hour and a half; in another series of 5 cases, after the ingestion of 15 grams, one woman recovered, and one woman and three men died, in a period of time varying from forty-five minutes to thirty hours. He found that 3/10 of a gram to 3 grams caused symptoms of poisoning, though the patients recovered; while, after a dose of 15 grams or more, 11 out of 12 patients died.

Professors Reid Hunt and Jones, in the latest book on toxicology (Peterson and Haines, 1904), state, referring to authorities, that death has occurred after as small a dose as 1.4 grams (22 gr.); and that a teaspoonful (3.7 c.c.) of the liquid acid proved fatal to a girl of seventeen, in twenty-three hours. According to the United States Pharmacopœia, 1 part of the pure acid crystallized at 15° C. (59° F.), is soluble in 15 parts of water. These same writers state, "on the other hand, recovery has frequently followed the taking of 1 ounce (31 c. c.)," which we assume refers to the saturated solution. "Two hundred and thirty-one and five tenths grains (15 gm.) may be taken as representing the minimum lethal dose." (Blyth). The German Pharmacopœia prescribes as a maximum medicinal dose, 0.7 grain (.045 gm.).

402. Treatment.—It should be remembered, from reading the foregoing pages, that carbolic acid acts as a poison in two ways: first, by the corrosive effect upon the mucous membrane which lines the entrances to the stomach and air passages; and, second, by an intoxication of the blood which produces narcosis and fatal action upon the brain and nerve centers. To counteract the first, attempts should be directed to removing the contents of the stomach, and to check the local action upon the mucous membrane of the mouth and throat. A pliable and soft stomach tube (flexible rubber) should be very cautiously introduced through the œsophagus into the stomach, and through this some saccharated limewater or a weak solution of sodium

sulphate should be used to wash out this organ; care should be used not to injure the lining of the gullet, because the acid may have softened its tissue. Sodium sulphate is a true chemical antidote, since, as we have previously stated, the pairing of carbolic acid with the sulphuric acid radicle forms phenylsulphuric acid, which is not a deleterious agent in the human economy. It should also be remembered that the local action upon the mucous surface, of causing a numbness of sensation or response to reflex action, would suggest that we should inject carefully a pretty fair amount of whatever liquid we have forced into the stomach, and then reverse the action by emptying the contents through the tube. External warmth should be applied to the surface of the body, and, after the stomach has been emptied, milk and demulcent drinks should be administered.

403. Action.—The action of this poison and the duration of its symptoms have been so sufficiently, in our opinion, portrayed in the foregoing pages as to require no further repetition.

404. Post-mortem appearances.—These are similar to the action of other corrosive and irritant poisons; the corrosive action usually extends from the mouth into and through the alimentary canal. The marks upon the lips, skin, and interior of the mouth, have been previously described; the peculiar white streaks and shriveled skin are very marked. The mucous membrane of the mouth and pharynx is reddened, and often edematous and swollen, sometimes it is whitened by the caustic action; but, as the local effects extend down the gullet and into the stomach, the mucous membrane becomes corrugated, bleached and loosened from the underlying tissues. In one of the cases above cited,[37] the mucous membrane of the stomach was almost entirely eroded, and "lay in lines along the posterior surface, looking and feeling like whipcord. The mucous membrane of the œsophagus was gathered into longitudinal folds, and presented very much the appearance of cigarette paper." Dr. Cameron[38] exhibited the stomach of a patient, which showed this rough, corrugated appearance of the mucous membrane of the gullet and stomach. In the Charing Cross Hospital case,[39] the root of the tongue and adjoining surface of the epiglottis presented a glazed appearance "of the color of recently cut lead," the surface of the gullet had a sticky feeling to the touch, and in its whole length had the same glazed, grayish look as the back of the tongue. This autopsy was made thirty-seven hours after death. The corrugated and shiny appearance of the gullet and stomach

[37] Brit. Med. Jour. April, 1881.

[38] Transactions of the Liverpool Med. Inst. July, 1881, p. 208.

[39] See *supra* § 395.

mucous membrane is commonly observed in this form of poisoning. In the lower part of the gullet and stomach, it may be detached in large pieces (as large as a silver dollar, sometimes), and these fragments have a dirty-red or greenish hue. The reddening of the mucous membrane ordinarily extends through the small intestine and into the large intestine. The liver is also congested and smells of the carbolic acid, and the same may be said of the kidneys. The blood is usually fluid, and has a peculiar, rich, mulberry color, and also smells of the acid. The lung tissue is usually edematous and injected. The brain is injected, and in one case[40] there was some slight excess of fluid in the ventricles, with a doubtful, faint odor of the acid; but in two cases[41] Dr. Allen reported that the brain was not much reddened, nor was the fluid between the membranes much increased.

The following condensed description of the pathological results of poisoning by carbolic acid is taken directly from the most recent textbook on pathology, and written by one of the highest authorities on pathological anatomy:[42] "Carbolic acid causes white or gray eschars in the mouth, which may be brownish from blood. In the stomach the eschars are not deep. The skin, where affected, is smooth and glistening. The odor is characteristic in the stomach, intestine, and other organs. Hemoglobinuria occurs, and pigment infarcts are found in the kidneys, and hyaline casts in the urine. The acid is excreted as alkaline salts of phenol-ether-sulphuric and phenol-glycuronic acids. The urine decomposes in the air and turns blackish."

The following detailed report of a carefully conducted autopsy after compound poisoning by carbolic acid, laudanum, and a soldering mixture, is taken from the medical examiners' returns in Massachusetts for the year 1889:

Case of Medical Examiner Draper, No. 106:

Ante-mortem symptoms: Vomited once; pressure at epigastrium; prostration; rapid pulse; stomach washed out; died from pneumonia on the third day after the poison was taken.

Post-mortem signs: Body emaciated. Heart normal. Lungs, left lower lobe darkened and, on section, edematous, engorged with several circumscribed nodules of solidification, adjacent to and surrounding bronchi; other parts of lung pale and normal. Spleen pale and small. Œsophagus, mucosa showed uniform, dark red color, most marked

[40] University College Hospital.

[41] Australian Med. Jour. March 15, 1880.

[42] A text-book on Pathology, etc., Hans Schmaus; translated by A. F. Thayer, M. D., edited by James Ewing, M. D.; Lea Bros. & Co., Phila. and New York, 1902, p. 362.

near its entrance to stomach. Stomach, not remarkable externally; mucosa, white, swollen at the pyloric outlet; the mucosa was of a dark olive-green color, swollen, with injection of submucous tissue; radiating from this were streaks of similar green color along summits of rugæ; at intervals between rugæ were limited areas of superficial ulceration. Duodenum and jejunum normal. Ileum, mucosa injected and reddened throughout its extent. Kidneys, cortex pale; and medulla red, with surface feeling granular to touch. Pancreas normal. Liver normal. Pelvic organs not remarkable. Meninges of brain, venous injection, brain substance moist and pale.

The same medical examiner reports, in 1898, to the same authority two autopsies on the bodies of two persons fatally poisoned, but the substance was not known. His return is as follows: Lips and tongue not seared, but pale. No external odor. Heart normal. Blood dark and fluid. Lungs congested. Spleen normal. Stomach, typical appearance of carbolic acid effect, namely, summit of rugæ whitened, with valleys between them much reddened and swollen; this organ contracted as if boiled, and emitted an odor of carbolic acid, on being opened. Pancreas normal. Intestines normal. Liver congested. Kidneys congested. Uterus enlarged, with signs of pregnancy. Ovaries normal. Bladder empty. Brain substance (head opened in one case only) wet, but not congested.

The above-quoted cases are so well described, and agree so exactly with the teachings of other well-recognized writings of toxicologists that it will be superfluous to enlarge this part of our subject.

405. Detection and tests.—Carbolic acid can be recognized very easily by the following tests: 1. Its peculiar odor resembling that of creasote. 2. The coagulation of albumen. 3. A pine splinter, soaked in an aqueous solution of carbolic acid, becomes blue when moistened with concentrated hydrochloric acid. 4. An aqueous solution of carbolic acid, when treated with a neutral solution of ferric chlorid or sulphate, becomes of a violet or beautiful blue color. 5. Lex's test consists in adding to a dilute solution of carbolic acid, one fourth of its volume of ammonia water, and then a little chlorid of lime (calcic hypochlorite) solution, when the mixture—especially after gentle warming—will become a beautiful blue color. This test is delicate for a solution containing 1 part of acid in 20,000 of water. 6. A solution of carbolic acid is precipitated by bromin water in the form of tribromphenol, a yellowish-white, flocculent precipitate. This reaction is made use of in estimating the amount of carbolic acid, the preciptate being collected upon a filter paper,

washed, dried, and weighed. One hundred parts of tribromphenol correspond to 28.4 parts of carbolic acid. 7. Another and exceedingly delicate test is by means of Millon's reagent, which produces, with dilute solutions of carbolic acid a beautiful red color, which is perceptible in solutions containing only 1 part of acid to 800,000 parts of water. 8. To a dilute, aqueous solution of carbolic acid that contains a trace of an alcoholic solution of ethyl nitrite is carefully added some concentrated sulphuric acid, in such a manner that the liquids do not mix. A rose-colored ring will form at the junction line. Delicacy: 1 part in 2,000,000 water.

Carbolic acid is isolated from the urine or other fluid by distilling it in a retort, after acidulating it with sulphuric or phosphoric acid. The distillates can be tested for carbolic acid by the above tests, and an aliquot portion of it precipitated with bromin water for the quantitative estimation. To analyze tissues for carbolic acid, they must first be finely cut up and extracted with very dilute sulphuric acid; after sufficient concentration by evaporation over a water bath, the residual fluid can be introduced into a retort and subjected to distillation as above mentioned. Carbolic acid can be separated from water by shaking the mixture with ether, pouring off the ether, after it has separated, into a watch glass or evaporating-dish, and allowing it to evaporate spontaneously, when the pure carbolic acid will be left as a residue.

IV. Formic acid.

406. In general.—Formic acid occurs in the acid secretion of red ants, in the blood, urine, bile, perspiration, and muscular fluid of man, and in the leaves of pine trees. It is produced in a number of reactions, by the oxidation of many organic substances, during the fermentation of diabetic urine and by the direct union of carbon monoxid and water. It is a colorless liquid with an acid taste and penetrating odor; it boils at 100° and crystallizes at 0°; it is miscible with water in all proportions.

Mineral acids decompose it into CO and H_2O, and oxidizing agents convert it into H_2O and CO_2. It acts as a reducing agent with the salts of silver, mercury, and other metals.

407. Symptoms of poisoning.—These resemble those of acetic acid; namely, coagulation of blood in the small vessels of the stomach, vomiting, abdominal pains, a staggering gait, convulsions, paralysis

of the lower extremities, dizziness, and weakness of the heart's action. When applied externally, it causes an urticarial eruption; when it is introduced hypodermically, as by means of the sting of a bee, hornet, or other insect (ant, formica), or by injury to the skin by certain hairy caterpillars (Lepidoptera, cabbage-white Pieris), the same itching eruption appears; animals eating this last-named insect may suffer gastritis, paralysis, etc. Formic acid is contained in the secretion, following the bites of these insects, and in a poison sac lying at the roots of the hair, in the others; this secretion contains formic acid. It is supposed that this acid is the cause of the cutaneous inflammation. The field nettle contains formic acid.

408. Action.—In addition to the coagulation of the blood, above mentioned, formic acid produces an alteration of the alkaline reaction of the blood, as in the case of acetic and other acids, and also changes its color to a bright red. It is eliminated by the kidneys, the urine voided having a brownish-red color.

409. Lethal dose.—This is said by Kobert to be less than that of acetic acid, the latter having a lethal dose of 308 grains (20 gm.).

410. Treatment.—Application of cold ice compresses for external injury; for internal administration, after a poisonous dose, emptying and washing out of the stomach, and the administration of chalk and magnesia, are recommended.

411. Post-mortem appearances.—These are, mainly, violet spots in the gullet, œsophagus, and stomach; the contents of the latter are stained brownish-black, and this may extend into the duodenum. Acid, grayish-brown masses are seen in the small intestines, and the peritoneum has a gray tinge. All the viscera exhibit the odor of formic acid.

412. Formaldehyd in general.—Formaldehyd, HCOH, is a colorless gas which has been used extensively as a germicide. It is produced by the dry distillation of calcium formate. It is used in medicine, in aqueous solution (40 per cent) called "formalin." It is very irritating to the mucous membrane of the nose and throat, and may cause severe conjunctivitis and bronchitis.

413. Detection and tests.—A solution of fuchsin that has been decolorized with SO_2 added to an aldehyd produces a red color. This is an aldehyd reaction and responds with formaldehyd.

Acidify formalin with sulphuric acid, add a few drops of dimethylanilin, and heat to 40° in a sealed tube for an hour or two. Make alkaline with sodium carbonate, and evaporate to excess of dimethyl-

anilin. Acidify with acetic acid, add a trace of lead dioxid, when a blue color will be produced if formaldehyd is present.[43]

A characteristic reaction is to add to formalin twice its volume of alcohol, and a few drops of diphenylenedihydrazin hydrochlorate. On warming, the solution is colored yellow and a yellow, crystalline precipitate is formed on standing.[44]

A little formalin heated with an equal volume of 40 per cent KOH containing 5 per cent resorcin gives a red color.[44a]

Dilute the material to be examined, with water, to a thin paste; acidify with sulphuric acid, and warm on the water bath, then add dry sodium sulphate in excess and distil. Formaldehyd will pass over in the first fractions, and may be tested as above.

V. Hydrocyanic acid and its salts.

414. Properties of hydrocyanic acid.—Hydrocyanic acid is a transparent, colorless, volatile liquid (b. p. 26.5°), with a pungent, acrid taste, which latter may be easily concealed in its administration in medicine or alcoholic beverages. It has a peculiar odor, often likened to that of bitter almonds or the kernels of peach stones.

415. Properties of oil of bitter almonds.—The oil of bitter almonds is about four times as strong as the medicinal hydrocyanic acid of the United States Pharmacopœia. It is of a yellow color, has a bitter, acrid, burning taste, and is slightly soluble in water. The almond flavors and essences, so much used in cooking, are solutions of the oil in spirit, and may prove highly dangerous in the hands of ignorant people. Enough prussic acid is contained in less than an ounce of most of these flavors, unless purified or exposed to the air for some time, to produce fatal effects, and it is evident that smaller quantities may have the same result in the case of children.

416. Properties of cyanid of potassium.—Potassium cyanid is a white, amorphous body, deliquescent, very soluble in water, with the odor of HCN, odorless when dry, insoluble in alcohol.

417. Cyanids of other metals in general.—In addition to hydrocyanic acid and potassium cyanid, the cyanids of other metals which are soluble in water, or easily decomposed by acids, are highly poisonous, such as sodium, ammonium, silver, mercury, and lead. In the last two, the effects of the metals are also often present.

[43] Trillat, Chem. Centralbl. 1898, II. p. 585.

[44] Neuberg, Ber. d. d. Chem. Ges. XXXII. p. 1962.

[44a] Chem. Centralbl. 1899, I. p. 641.

418. Oil of bitter almonds in general.—Hydrocyanic acid may be obtained from many vegetables, particularly those belonging to the suborders Amygdaleæ and Pomeæ;[45] as from bitter almonds, apple pips, the kernels of peaches, apricots, cherries, plums, and the flowers of the peach, and cherry laurel, from the bark of the wild cherry, and the root of the mountain ash. Prussic acid does not exist ready formed in these plants, but is the result of the reaction of water upon amygdalin in the presence of a ferment, emulsin. Hence, if any of the above substances are found in the stomach, the question may arise whether the indications of the presence of prussic acid are due to their decomposition or to the acid swallowed as such. The only manner in which doubt, arising from this circumstance, can be satisfied, is the obtaining by chemical analysis a larger quantity of the acid from the contents of the stomach than these substances could yield. It is extremely improbable that death could result from the ingestion of any of these articles, except in such a large quantity as to obviously preclude the idea of prussic acid, in substance, having been taken.

It has been stated that this acid may be produced spontaneously from unsound cheese; but Dr. Taylor was unable, by experiment upon numerous samples of decayed cheese, to find any evidence of it. The notion, also, that it may be a spontaneous product of animal decomposition, is timidly advanced by Orfila, but has not yet received the necessary confirmation. It is also said to be produced by the action of nitric acid on alcohol. This fact was clearly ascertained by M. de Claubry, who observed the serious effects of the vapor upon the health of the workmen engaged in the manufacture of hyponitrous ether.[46]

"The following plants with many others, all yield, by appropriate treatment, more or less prussic acid: Bitter almonds (amygdalus communis); the amygdalus persica; the cherry laurel (Prunus Laurocerasus); the kernels of the plum (Prunus domestica); the bark, leaves, flowers, and fruit of the wild service tree (Prunus Padus); the kernels of the common cherry and apple; the leaves of the Prunus capricida; the bark of the Prunus Virginiana; the flowers and kernels of the Prunus spinosa; the leaves of the Cerasus acida; the bark and almost all parts of the Sorbus aucuparia, S. hybrida, and S. torminalis; the young twigs of the Cratægus Oxyacantha; the leaves and partly, also, the flowers of the shrubby

[45] Pereira. [46] Ann. d'Hygiène, 1839, II. p. 350.

Spiræeæ, such as Spiræa Aruncus, S. sorbifolia, and S. Japonica; together with the roots of the bitter and sweet cassava.

"In only a few of these, however, has the exact amount of either prussic acid or amygdalin been determined; 1 gram of bitter almond pulp is about equal to 2½ milligrams of anhydrous prussic acid. The kernels from the stones of the cherry, according to Geiseler, yield 3 per cent of amygdalin; therefore, 1 gram equals 1.7 milligrams of (HCN) anhydrous acid.

"The wild service tree (Prunus Padus) and the cherry laurel (Prunus Laurocerasus) contain not amygdalin, but a compound of amygdalin with amygdalic acid; to this has been given the name of laurocerasin. It was formerly known as amorphous amygdalin; its formula is $C_{40}H_{55}NO_{24}$; 933 parts are equivalent to 27 of hydric cyanid, that is, 100 parts equal to 2.89 hydric cyanid.

"In the bark of the service tree, Lehmann found .7 per cent of laurocerasin (=.02 HCN), and in the leaves of the cherry laurel 1.38 per cent (= 0.39 HCN).

"Francis, in a research on the prussic acid in cassava root, gives as the mean in the sweet cassava .0168 per cent, in the bitter .0275 per cent; the maximum in each being, respectively, .0238 per cent and .0442 per cent. The bitter, fresh cassava root has long been known as a very dangerous poison; but the sweet has hitherto been considered harmless, although it is evident that it also contains a considerable quantity of prussic acid.

"The kernels of the peach contain about 2.85 per cent amygdalin (= .17 HCN); those of the plum .96 per cent (= .056 HCN); and apple pips .6 per cent (= .035 HCN).

"It is of great practical value to know, even approximately, the quantity of prussic acid contained in the various fruits, since it has been adopted as a defense in criminal cases, that the deceased was poisoned by prussic acid developed in substances eaten." (Blyth.)

The essential oil of bitter almonds, which is the product of the distillation of the crushed almonds with water, contains a large amount of prussic or hydrocyanic acid, which is the product of the chemical action of the ferment emulsin upon the glucosid amygdalin, both of which pre-exist in the almond pulp, but do not yield prussic acid until brought together in contact with water. It has been estimated that 2,500 parts of almond pulp contain 100 parts of amygdalin, and yield, by distillation, 41 parts of essential oil, which contains, when fresh, 6 parts of anhydrous prussic acid. Hence the essential oil may, when perfectly fresh, contain 12.76 per cent of

anhydrous acid. This, on account of its volatility, escapes very rapidly, if the oil be exposed to the air, and a portion escapes every time the bottle is opened, as is the case with all preparations containing hydrocyanic acid.

It is, therefore, a dangerous substance, from the very fact of the uncertainty of the amount of the contained poison. In certain patent medicines made from wild cherry, the hydrocyanic acid present may reach a dangerous amount. Heat, used in cooking, may volatilize a considerable portion of the prussic acid in the bitter almond oil, and hence secure a certain immunity from its baneful influences, but when used without heat, caution should be exercised. It should be remarked here, however, that the peculiar flavor and taste do not depend upon the amount of prussic acid present, since a distillation of the oil in the presence of potassic hydrate will remove the prussic acid, and without dispelling the almond flavor. Even with this precaution the essential oil, like all other essential oils, will cause an intoxication like that of alcohol. There has been, at times, some doubt expressed as to the fact of this purification being sufficient. Much of the difference of opinion, which has reigned as to this question, arises from the circumstance, that by merely agitating the oil with a large excess of lime or caustic potash, and distilling it, the prussic acid is not sufficiently separated. A salt of iron should be employed, which fixes it more effectually. Dr. Maclagan made experiments upon dogs with the oil thus rectified, and found that, when no trace of prussic acid could be detected by the iron test, it was not poisonous. The following are the most prominent of his conclusions: 1. That the poisonous action of the unrectified oil is essentially due to the hydrocyanic acid which it contains. 2. That the oil really free from prussic acid, in doses of a few drops, does not act as a poison on animals generally. 3. That although the rectified oil, in doses of a dram and upwards, does prove fatal to rabbits, yet in dogs (whose organization renders them much better subjects for testing the probable effects of the substance on man), doses of the oil even as large as 3 drams, entirely or nearly free from prussic acid, produce no other effect that a little vomiting, and do not cause death, or even dangerous symptoms. The same results have, in the main, been obtained by other chemists, particularly Wöhler and Frerichs.

Why the deadly ingredient should be allowed to remain in it, if it can be so readily removed, it is not easy to understand. The placing of restrictions upon the sale of this, and other articles of a pernicious character, for the purpose of flavoring or ornamenting

articles of food, appears to us to be of still more urgent importance than any restraint upon the sale of arsenic and similar poisons, which cannot be employed except for destructive purposes. In the latter case, means are readily found to obtain the required poison; while in the former, experience has shown that equal brilliancy of color and delicacy of flavor can be obtained from harmless substances, as from the deadly poisons in universal and daily use in this country and England. Prussic acid is too potent a poison to be distributed to cooks and confectioners, disguised with the pleasant odor of bitter almonds; the most ordinary prudence and humanity would seem to demand that it should not be used thus freely and incautiously.

419. **Preparations.**—The medicinal or dilute acid (U. S. P.) contains 2 per cent of anhydrous acid. The dilute acid (Br. P.) has about the same strength, but Scheele's solution of this acid contains 5 per cent or less of the anhydrous form. The U. S. P. directs that the 2 per cent by weight of absolute hydrocyanic acid, and 98 per cent of water shall be made in the following manner:

Potassium ferrocyanid, in coarse powder, twenty grams	20 parts.
Sulphuric acid, eight cubic centimeters	8 "
Water	65 "
Distilled water, a sufficient quantity to make a 2 per cent solution.	

Potassium cyanid, silver cyanid, gold cyanid, and amygdalin, are all considered to possess the poisonous qualities of hydrocyanic acid, which they contain; potassium cyanid especially, when swallowed into the stomach, is probably decomposed into another potash salt, giving off slowly, if the stomach is full of food, hydrocyanic acid, which is absorbed as such into the system; if we except the supposition that this cyanid salt may be decomposed in the mouth in part, and so offer the means of its inhalation by the lungs, this slow decomposition may explain why death does not occur so rapidly, when this salt is swallowed, as when a dilute solution of hydrocyanic acid is swallowed.

420. **Statistics of poisoning from use of hydrocyanic acid.**—By reference to Mr. Blyth's table, as given in the early part of chapter I., it will be noticed that, during ten years ending 1892, the number of deaths occurring from ingestion of prussic acid and its cyanids amounted to 426; 14 of this number were homicides or murders, 344 suicides, 68 by accident or negligence. Referring to the table of the returns of the Massachusetts medical examiners for the period of twenty-five years, it will be noticed that there were 75 deaths by this

acid and its cyanids, of which 2 were murder, 68 were suicides, and 5 by accident or negligence. (pp. 18–20.)

Accidents have occurred from errors in dispensing, among which the following tragic result is recorded:[47] An apothecary substituted, in a prescription, cyanid of potash for chlorate of potash, and the child died after the first dose; being sure that he had made no mistake, the apothecary drank some from the same bottle and immediately died; Dr. Arnold himself also took of the bottle, became unconscious for six hours, and then recovered.

Mr. Blyth,[48] referring to a collation of twelve years' statistics by Falck, gives the following results:

Fatal cases of poisoning by potassic cyanid	29
" " " " " hydric cyanid	9
" " " " " oil of bitter almonds	5
" " " " " peach stones	3
" " " " " bitter almonds	3
" " " " " tartaric acid and potassic cyanid	1
" " " " " ferrocyanid of potassium and tartaric acid	1
Total	51

Peterson and Haines give the percentage of mortality out of 455 cases of cyanid poisoning, many of which were of slight nature, as 84; and in another series of 40 cases the mortality was 95 per cent.[49]

421. Symptoms of poisoning.—The rapidity with which this poison acts upon the system hardly allows of the observation of successive symptoms. Where insensibility is not immediately produced, it is preceded by faintness, giddiness, loss of muscular power, and sometimes by convulsions. In other cases, the patient being found insensible, it is impossible to know the previous symptoms. When seen at this time, the eyes are fixed and glistening, the pupils dilated and unaffected by light, the limbs flaccid, the skin cold and covered with a clammy perspiration; there is convulsive respiration at long intervals, between which the patient appears lifeless; the pulse is imperceptible, and involuntary evacuations occasionally take place; the respiration is slow, deep, gasping, and sometimes heaving or sobbing. This description, which is applicable to the greater number of cases, we have borrowed from Dr. Taylor. It should be added,

[47] A. B. Arnold, "Case of poisoning by the cyanid of potassium," Am. Jour. of Med. Sci. 1869.

[48] Op. cit. p. 197.

[49] Op. cit. p. 577.

that there is usually rigidity of the jaws, which has sometimes effectually prevented the administration of antidotes.

The period at which death takes place is usually within an hour, seldom indeed exceeding three quarters of an hour. A man drank from a vial containing prussic acid while embracing his wife; he died in fifteen minutes.[50] Seven epileptic patients, who were accidentally poisoned with this acid, died in convulsions within three quarters of an hour.[51] In most cases, however, death occurs in a few minutes; and, if life be prolonged for a period of an hour (Lonsdale), recovery usually takes place.[52] The rapidly fatal character of this poison is, indeed, one of its most striking features. From experiments upon animals, it was supposed at one time that prussic acid was, necessarily, almost immediately fatal. Animals, poisoned by it, die within a few seconds. In man, however, although the symptoms often commence in the act of swallowing, either by direct absorption through the surfaces of the tongue and mouth, or more probably, by inhalation and absorption through the lungs, they may also not be perceived for one or two minutes. When absorption occurs by inhalation in an atmosphere containing a small amount of the volatile acid, whether over the fumes from a vessel in the open air, or in a room badly ventilated, the symptoms of intoxication may come on at a later period. The danger of accident will depend upon the amount at any one period present in the system of the individual exposed to its vapor or solution, and if this be sufficient to imperil his life, the symptoms will occur suddenly and with great violence.

As above remarked the appearance of symptoms of hydrocyanic poisoning will depend not only upon the size of the dose and the strength of the solution swallowed, but also upon whether the stomach contains food or is empty. When a large amount of this acid is swallowed, the poisonous effect begins within a few seconds to one or two minutes. The peculiarity of respiration after the absorption of the poison is very striking; there is a shortening of the ordinary period of inspiration, and a prolonged, most often forcible, period of expiration. The cause of this peculiar mode of respiration, which has been likened to the Cheyne-Stokes respiration peculiar to cerebral diseases, will be described later on, when we speak of the action of this poison. (§ 427.)

On account of the very rapid appearance of these poisonous symp-

[50] Pharm. Jour. Aug. 1851.
[51] Orfila, Vol. II. 286.
[52] Dr. Fagge reports one fatal case which occurred in an hour and a quarter after the poison was swallowed. Guy's Hosp. Rep. 1868, p. 259.

toms, an important question arises bearing upon the voluntary or homicidal nature of the poisoning, since it may become evident, from circumstantial evidence, that the deceased has retained consciousness and voluntary power for a certain length of time after swallowing the poison. In Mr. Burman's case, mentioned further on, insensibility did not occur for two minutes after the poison was swallowed, and that in the largest dose yet recorded. In the case of a girl, aged seventeen, the servant of a chemist, who was seen by the reporter when already insensible, the retention of consciousness for a short period was proved by circumstantial evidence. In turning up the feather bed, after the body had been removed from it, a prussic acid bottle, with the stopper in, was found between it and the mattress, near the center; it contained about eight drops of the acid. The girl's mistress stated that, about twenty minutes after the girl had left her, she was proceeding up stairs to bed, when, in passing the girl's room door, she heard a moaning noise; she entered the room, and found her lying in bed, with her clothes on, and the bedclothes drawn up to her face, apparently "gasping for breath." She instantly gave the alarm. "The evidence adduced proved, as far as could be proved, that she had swallowed an ounce of the acid, recorked the vial, thrust it to full arm's length between the feather bed and the mattress, got into bed, and then drew the clothes over her body; and there appeared to have been no convulsions."[53]

Dr. Sewell reports, in the same journal, the interesting account of a gentleman who swallowed 7 drams of the medicinal acid, equal to 21 grains of Scheele's acid. It was proved that, after taking the poison, he had walked from the table in the middle of the room to the door, unlocked it, called for assistance, and then, returning to a sofa in the room, stretched himself upon it. Here he was found lying as if in a profound slumber, his legs crossed, his arms by his side, and his eyelids firmly closed. The eyes were more brilliant than during life, and continued so until the next day. His face was livid, and the lips were blue; the muscles were all relaxed.[54]

A young man swallowed, in his bedroom, a dose equivalent to 2.54 grains (0.17 gm.) of anhydrous acid. He then descended thirty steps, and walked about twenty paces before he became powerless. He was endeavoring to open the front door of the house to go out, when he suddenly fell. The only symptoms observed by a person

[53] Boston Med. and Surg. Jour. Vol. XXXII. p. 528; Leithead.

[54] *Ibid.* Vol. XXXVII. p. 320.

present were that "he threw his arms about, and made a noise in breathing, fetching it hard; he very soon became still." [55]

A case, which is characterized by Dr. Taylor as one of the most extraordinary on record in this respect, is that related by Mr. Godfrey: "A gentleman, aged forty-four, swallowed, it was supposed, half an ounce of prussic acid (strength not stated), but certainly a quantity sufficient to destroy life. After taking it from the bottle, he walked ten paces to the top of a flight of steps, descended the stairs, seventeen in number, and went to a druggist's shop at forty paces' distance, where he had previously bought the poison, entered the shop, and said, in his usual tone of voice, 'I want some more of that prussic acid!' He then became insensible, and died in from five to ten minutes after taking the poison." There were no convulsions.

Such cases as these (and more might be quoted) fairly prove the untenable nature of the notion that any acts indicative of design, committed after the poison has been swallowed, cannot be attributed to the deceased. Many simple acts, like those noted, can be easily performed in a very short space of time, and scarcely take anything away from the fearfully rapid character of this poison.

Another fallacy, derived from the result of experiments on animals, is the supposition that death from prussic acid is always preceded by a shriek. There are few, if any, cases in the human subject which attest any such fact; on the contrary, in the vast majority of cases, there are neither general convulsions, as is common in animals, nor any unusual cry; but death comes on in a placid manner, the patient passing away without a struggle. The convulsions which were observed in the seven epileptic patients already referred to, may, with some probability, be referred to their constitutional predisposition. In a case of suicide by a dose equivalent to 8 grains (reported by Dr. J. G. Fleming), the appearance of the body was most strikingly like life, even the natural color had not left the cheek, the features were composed, and the limbs relaxed. There evidently could have been no convulsions.[56]

The following symptoms of fatal poisoning from a three-dram dose of prussic acid are reported by Dr. A. B. Kelly:[56a] Perfect insensibility, lividity, mucus about the mouth, eyes open and glassy, pupils dilated, respiration hurried and stertorous, no convulsions or vomiting. Opthalmoscope, fifteen to twenty minutes after dose was taken, showed that the brilliant red glow of the choroid was entirely absent,

[55] Lowe, Guy's Hosp. Rep. 1846, p. 490.
[56] Edin. Monthly Jour. July, 1846.
[56a] Lancet, Dec. 6, 1879, p. 831.

a pale violet-gray tint taking its place. The optic disk, of a dull gray-white tone, was ill defined; the retinal arteries were discerned with great difficulty, as narrow threads; the veins, on the other hand, were easily traced, appearing unevenly and imperfectly charged with blood; the media presented the appearance of baked tapioca. These appearances were observed after artificial respiration, and faradization of the phrenic nerve, were resorted to, and were indicative of death.

422. **Action.**—This poison is also one which disturbs oxyhemoglobin, held in feeble combination in the blood corpuscles. Early investigations upon this question by Preyer,[57] Hoppe-Seyler,[58] Bernard,[59] Meuriot,[60] Wagner,[61] and Lewisson,[62] are quite fully set forth in a publication on materia medica and toxicology,[63] and show very conclusively that the action of this poison is direct, by its presence in the blood, upon the organic life, producing a similar and as fatal a result as if the animal life had ceased by suffocation. When the doses taken are small, nontoxic doses, their continuous administration causes a slowing of the heart's action, following almost immediately upon a sudden, prolonged arrest of the heart in diastole (or relaxed in opposition to a contracted state of that organ), which may finally result in the complete arrest of cardiac movements. In certain experiments of Boehm and Knie,[64] it was observed that if woorali were first given to animals, the injection of prussic acid could be well supported in large doses, provided artificial respiration were maintained.

Whether the absorption of the poison is accomplished by the inhalation of its vapor, or whether the solution of the vapor of the hydrocyanic acid in water be received into the stomach, the rapid appearance of these peculiar symptoms is explained by the more recent toxicologists as due to direct intoxication of the blood. This volatile, gaseous agent, received into the circulation of the blood, forms a more stable combination with the hemoglobin of the blood corpuscle, thus displacing the less stable oxygen combination previously existing. The combination of the cyanogen gas compound makes a new com-

[57] Die Blausäure, Bonn. 1870.

[58] Medicinisch-chemische Untersuchungen, Berlin, 1867.

[59] Leçons sur les substances toxiques des medicamenteuses, Paris, 1857.

[60] Archives Générales 6e s. t. XI.

[61] Lancet, 1877.

[62] Reichert's Archiv. 1870.

[63] H. C. Wood, Phila. 1882, 4th ed.

[64] Archiv. für exper. Path. und Therap. Bd. II. p. 137, referred to by Prof. Wood, op. cit. It is not at all improbable that the presence of the arrow poison (woorali) in the blood may prevent the blood corpuscles absorbing the more volatile prussic acid, and the latter be eliminated with exhalation from lungs. Editor.

bination called cyanmethemoglobin, which imparts a bright red color to the blood; the presence of this cyan combination prevents the blood corpuscles from imparting oxidation to the tissues, and the interchange between the blood and the tissues (metabolism) ceases; the functions of the organs of the body, therefore, cease during this tissue intoxication, and the above, poisonous symptoms are the effects. These results are difficulty of respiration, weakness of the heart action, contraction and spasm of the muscles, suspension of cerebral functions in the nerve centers, spasmodic contraction of the involuntary muscular fibers, a stasis of the circulation of blood in the smaller veins and capillary system of vessels, and a suffusion of liquid blood in the right side of the heart, with an engorgement of the pulmonary blood vessels.

Many writers have spoken of the more tardy appearance of symptoms of poisoning by this agent, when the cyanid of potash or other poisonous cyanid salts have been taken into the stomach; this has been explained on the supposition that the evolution of hydrocyanic acid, resulting from the decomposition of its association with the potash, is accomplished slowly, and, in consequence, small, continuous amounts of the acid pass into the blood circulation. The following interesting case of poisoning by a large dose of cyanid of potash, and a recovery, affords a possible explanation why an ordinarily fatal dose did not cause death. This case is reported in the London Lancet for December 18, 1886, as follows:—

Sophia P., aged thirty, swallowed a piece of cyanid of potash, with the intent to commit suicide, and almost immediately became unconscious and was taken to the hospital, where she arrived in fifteen minutes after the dose was taken. The amount which she swallowed was as big as a lump of sugar; artificial respiration was continued for two hours, and, during this period, she passed urine which smelled strongly of prussic acid. The stomach pump was used to empty the stomach of its contents, which were replaced with a solution of ferrous sulphate to form the insoluble precipitate of Prussian blue; this was immediately pumped out again for the purpose of removing any unabsorbed cyanid salt. Simultaneously with these methods of treatment, two subcutaneous injections of sulphuric ether were given, at intervals of two hours. This patient made a complete recovery.

The above case would appear to parallel an experiment, which the writer used to exhibit before his medical class, to illustrate the method of controlling the absorption of hydrocyanic acid by means of ether inhalation. This form of experimentation shows that filling

the blood and the tissues of the body full of ether, in an animal exposed to its inhalation, will retard the poisonous action of hydrocyanic acid sufficiently for the animal to recover from etherization, and to be allowed to breathe fresh air. It will be noticed that, in the above-recited case, the stomach pump was used to empty the stomach of its contents, and a ferrous sulphate given to form Prussian blue, which was also removed; afterwards ether was administered by subcutaneous injection. The sudden absorption by the blood corpuscles, of the innocuous, etherous vapor saturation, would prevent the prussic acid from forming a poisonous combination with the corpuscles; and by pumping out the decomposed cyanid salt from the stomach, and the elimination of the poison by the eliminating organs of the body, this drug was removed from the system. This fact of elimination is especially proved by the unmistakable, strong smell of the prussic acid in the urine of Sophia P., which the kidneys had first removed from the blood and afterwards excreted into the bladder and so passed out.

Dr. Taylor[65] discusses, as an important medico-legal question, the rapidity with which death may occur after a fatal dose of prussic or hydrocyanic acid. Since its action is so rapid, it becomes important to know for how long a time the individual was conscious and able to move of his own volition. Dr. Lonsdale is the authority for the statement that 1 dram of Scheele's acid (equal to about 2½ drams of the dilute acid of the U. S. P.) would affect the ordinary adult in one minute or less, and three or four times this dose will exert its influence within ten or fifteen seconds. Dr. Taylor cites the importance of this question in the case of *Rex* v. *Freeman* at the Leicester spring assizes, 1829.[66-7] "The medical question at the trial was: Could this quantity of poison, 4½ drams of Scheele's solution, equal to about 11 fluidrams (about 40 c.c.) of a dilute hydrocyanic acid solution of the U. S. P., have been taken, and the deceased have retained consciousness and volition for a sufficiently long period to have performed these acts herself (corking the vial and placing the leather and string, which appeared to have gone round the neck of the bottle, in the chamber vessel).[66-7] Five medical witnesses were examined and the opinions of four of these were strongly against the possibility of the acts having been performed by the deceased. This strong medical opinion was set aside by circumstances and the prisoner was acquitted." We can see no reason for the assumption that

[65] Treatise on Poisons, Phila. 1873, p. 565.

[66-7] Medical Gazette, Vol. VIII. p. 759.

the deceased must have placed the articles spoken of in the vessel after swallowing the poison. Why would it not be more reasonable to assume that this act of volition was performed before swallowing the prussic acid? Two other cases are referred to by Dr. Taylor, in which the individuals, who took the acid for suicide, used similar, large doses and set the vials down by their side, drew up the bed-clothes smoothly without any signs of disorder about them or the body itself. Dr. Taylor states that "in death from prussic acid the body is usually found lying calm and tranquil, without any mark of struggling or convulsions."

423. **Diseases which simulate symptoms of hydrocyanic acid poisoning.**—The diseases which are liable to be confounded with poisoning by this acid are sudden apoplexy, cerebral or pulmonary, cardiac syncope, embolism of the lungs, or air embolism in the arteries or veins after childbirth or instrumental abortion. The question of doubt in these cases can always be solved by a careful post-mortem examination, or by an early, chemical examination after death,—especially of the stomach contents.

424. **Symptoms of chronic poisoning.**—These are said to occur with persons working with photographic materials and in gilding, by the handling of the preparations of the cyanid compounds. The symptoms observed are headache, dizziness, noises in the ears, troubled respiration, pain over the region of the heart, a constriction of the muscles of the throat, inappetence, nausea, obstinate constipation, a full pulse with pallor and offensive breath. Koritschoner[68] observed the symptoms of patients, breathing, at intervals, during many weeks, hydrocyanic acid vapor. This treatment he intended for the destruction of tubercle bacilli. "Twenty-five per cent of these patients suffered from redness of the pharynx, salivation, headache, nausea, vomiting, slow pulse, and even albuminuria."

425. **Diagnosis.**—From the foregoing description of the action and of the symptoms of hydrocyanic acid poison, it may be easily inferred that the history of a very rapid death (one to three minutes) would suggest the kind of poison taken; but it should be remembered that there are a few other poisons producing death almost as quickly; for instance, carbolic acid and oxalic acid. Most of the substances which cause so rapid a death can be recognized from the odor belonging to the poison. The history of the symptoms, which would undoubtedly be related by any bystander, of the peculiar, respiratory movements, the staring expression of the eyes, the rigidity of the muscles of the

[68] Wiener klin. Woch. 1891.

jaw, and the involuntary discharge from the bowels, etc., would also help in the diagnosis. The odor of hydrocyanic vapor would be observed; but this might be masked by some other substance having a stronger odor, and thus would tend to obscure the peculiar odor of hydrocyanic acid. The symptoms of poisoning by hydrocyanic acid may be confounded, as reported by Cox, where, evidently, the poison taken was oil of mirbane (nitrobenzol), known as artificial oil of bitter almonds; but this poison does not usually show symptoms under fifteen minutes to three hours after taking the poison. With this artificial oil of bitter almonds the skin has a grayish or bluish-white complexion, the lips, and nails on the fingers, being purple.

426. **Treatment.**—When a solution of this acid has been swallowed, the rapidity of its effects is a bar to many remedial measures; the indications are to prevent the absorption of the acid into the blood, to accomplish which, artificial respiration should be attempted. If there were time, ether might be given for inhalation or by injection subcutaneously to delay further absorption of poison, as in case of Sophia P. (§ 422). If oxygen gas can be obtained, this should be inhaled into the lungs; a ferrous sulphate solution may be pumped into the stomach to precipitate any unabsorbed poison in the form of Prussian blue, and the stomach washed out immediately afterward. When a solution of the salts, cyanid of potash or other cyanid salts, has been swallowed either in bulk or solution, the use of the stomach pump and the ferrous sulphate salt, as well as a hydrated oxid of iron solution (U. S. P.), which is usually kept by druggists for such emergencies, should be administered, and the stomach washed out. Kobert suggests that hydrogen peroxid be given in solution by introduction into the stomach, or injected subcutaneously; he also advises the use of potassium permanganate. "From experiments made by Krohl[69] on dogs and cats, hydrogen peroxid appears to be an antidote to hydrocyanic acid, the apparent action being the oxidation, by the hydrogen peroxid, of the hydrocyanic acid to oxamid. These experiments show that hydrocyanic acid in larger quantities than the fatal dose, when administered to dogs and cats, had its effects stayed by means of hydrogen peroxid, and, moreover, the hydrocyanic acid could not be detected in the urine after the employment of hydrogen peroxid, whereas it is present in ordinary cases of hydrocyanic poisoning."

427. **Lethal dose of hydrocyanic acid.**—The fatal dose of hydrocy-

[69] Arb. Pharmakol. Inst. Dorpat, 1892.

anic acid will depend very much upon the strength of its solution in water. While the required strength of the Pharmacopœia, both in England and in America, should contain 2 per cent of the anhydrous acid, and Scheele's solution 5 per cent, it is more likely that these required strengths—especially if the bottle containing the solution should be kept in the light, and for a long time—will be less than indicated. The smallest dose we have been able to find,[70] which has been followed by death, is 30 minims (1.87 c.c.) of Br. P. solution of the dilute acid, which is equal to .6 of a grain (0.04 gm.) of the anhydrous acid. Recovery has occurred after a dose of 4 drams of the Br. P. acid,[71] which is equal to 4.8 grains (0.308 gm.) of the anhydrous acid; but in this case the stomach was quickly washed out and other energetic treatment followed. In still another case,[72] a dose of two teaspoonfuls of the dilute acid, equal to 2.5 gr. (.16 gm.) of the anhydrous acid, was taken, and the woman recovered. The treatment by the immediate use of stomach pump, and subcutaneous injection of apomorphin, with the employment of electrical stimulation and artificial respiration, was prompt. These cases are quoted in the more recent books on toxicology. The shortest time in which hydrocyanic acid has proved fatal is two minutes, and the longest an hour and a half.

The amount which destroyed the life of a woman, mentioned by Mr. Hicks,[73] was 0.9 gr. (.058 gm.). Life was extinct in twenty minutes. This quantity is equal to about 30 drops of a two per cent solution of the acid. Other cases have been reported in which most alarming symptoms ensued from smaller doses.[74] In any case in which it appears that death has resulted from a small quantity, it is highly important, if we would avoid errors, that the strength of anhydric acid should be ascertained by an analysis of the sample remaining.

Instances of recovery from very large doses have been recorded. Dr. Christison has reported a case in which, with great difficulty, a gentleman was restored, who had taken between 1½ and 2 gr. (.096 to .13 gm.) anhydrous acid;[75] and, four years later, Mr. W. H. Burnam communicated to the Lancet a very interesting history of the recovery of his father from accidentally taking a dram of Scheele's acid, which was found upon analysis to contain 2.4 grains (0.156 gm.) of

[70] Garstang, Lancet, 1888, Vol. II. p. 15.
[71] Internat. Jour. Med. Sci 1890.
[72] Brit. Med. Jour. 1890.
[73] London Med. Gazette, XXXV. 896.
[74] *Vide* Taylor on Poisons.
[75] Med. Gazette, 1850, 917.

anhydrous acid.[76] Mr. Nunnely, also, has reported a case of recovery from 1⅓ grains (0.087 gm.) of anhydrous acid.[77] One curious fact, in relation to the size of the dose, should not be forgotten, *viz.*, that a comparatively small dose will produce equally fatal results with a large one, it being highly probable, from the cases so far recorded, that all doses over 1 grain (0.065 gm.) of absolute or anhydrous acid are capable of destroying life with equal certainty and rapidity. The limits of safety in the use of prussic acid are easily passed, and the formidable symptoms occasioned by it develop themselves with wonderful rapidity; hence, too great caution cannot be observed in its administration with remedial views in medical practice.

428. Other methods of receiving hydrocyanic acid into the system than by swallowing.—M. Regnauld relates the case of a young man who was poisoned by the vapor of prussic acid, disengaged from a mixture of the ferrocyanid of potassium and sulphuric acid. This is the prescribed formula for making dilute hydrocyanic acid, U. S. P. The symptoms were those of asphyxia, rather than of the nervous prostration, which usually accompanies poisoning from the internal use of prussic acid.[78]

It is a matter of history that the noted chemist Scheele, the discoverer of hydrocyanic acid, died by inhaling the vapor of this acid from a broken flask. There is also the historical case cited by Prof. Tardieu, in which a case of poisoning from this acid occurred by its introduction under the finger nails, and another case mentioned by Carriere[79] in which a woman attempted suicide by taking an enema containing cyanid of potash. M. Grehant[80] has shown by experiments, in which every precaution was used to prevent inhalation of fumes, that hydrocyanic acid applied to the eyes of warm-blooded animals may destroy life in a few moments.

429. Fatal use of bitter almonds.—Mr. Blyth, assuming the dictum of Taylor, *viz.*, that an equivalent to one English grain (65 mg.) of the anhydrous acid would, under ordinary circumstances, be sufficient to destroy adult life, makes the following calculation: About 80 bitter almonds contain 2.5 per cent of amygdalin, and, as amygdalin yields 5.9 per cent of hydrocyanic acid, 80 bitter almonds should, theoretically, be a poisonous dose for an adult; with children less than this number; in fact, 4 to 6 bitter almonds are said to have

[76] Brit. and For. Med.-Chir. Rev. April, 1854.

[77] Taylor.

[78] Brit. and For. Med.-Chir. Rev. Oct. 1852. p. 561.

[79] "Empoisonnement per le cyanure de potassium.-guérison" Bullet. general de Thérap., 1869, No. 30.

[80] N. Grehant, Compt. rend. Doc. Biol. (9) XI. 64, 65.

produced poisoning in a child. Baker reports a case[81] in which a man stated that he had eaten two handfuls of bitter almonds; in spite of severe symptoms he recovered.

It should be remembered that the decomposition of prussic acid from the amount of almonds swallowed would be performed slowly, so that, during the process of its evolution and absorption in the animal economy, much of the poison would be slowly eliminated.

A lad of fifteen, the son of a wholesale grocer, got access to a cask of bitter almonds, and consumed a large quantity of them with sugar. After a time, but how long is not known, he felt a pleasing sensation, then became suddenly giddy, fell down, and lost his consciousness and recollection. He was found lying insensible near the cask. Ammonia and carbonate of potassium were successively administered, and the stomach pump employed. By these means he was much relieved. Emetics were then given, and he threw off, in the course of half an hour, as much as 8 ounces, Troy, of bitter almonds.

Another case of poisoning,[82] from eating two handfuls of bitter almonds, is reported, in which the first symptom observed was a convulsion with gasping and labored breathing. The patient recovered after the application of electricity, one pole being applied over the heart, the other over the course of the pneumogastric nerve in the neck. The contents of the stomach were first removed with the aid of a stomach pump. Inhalations of ammonia, beef tea, and brandy were also administered. The ammonia inhalations and electricity were applied alternately every half hour.

It should be noted that, though Prussian blue, Turnbull's blue, etc., which are double cyanids, are considered nonpoisonous, yet when these are taken simultaneously with acids, hydrocyanic acid may be set free in the stomach, and cause poisoning. It may be noted in Mr. Blyth's table, above mentioned, that there was one fatal case of poisoning caused by taking a mixture of ferrocyanid of potassium and tartaric acid. This is not strange, as, by the formula of the U. S. P., dilute hydrocyanic acid is made from these materials, sulphuric acid being substituted for tartaric.

430. Fatal use of oil of bitter almonds.—The following are a few of the instances in which the oil of bitter almonds has produced fatal results:—

A child, eight and a half years old, took a teaspoonful of ratafia,

[81] Brit. Med. Jour. 1881, Vol. II. p. 12.
[82] Med. Times and Gazette, London, July 9, 1881.

containing 7 drops of the oil of bitter almonds. She became immediately insensible, but had no spasms; the limbs were relaxed, the jaw, however, firmly closed; the eyelids closed, but the eyes were brilliant and glassy, although without expression. Cold affusion, emetics, and stimulants restored her, and in twenty minutes her consciousness returned.[83] The general symptoms of poisoning with the oil of bitter almonds resemble very closely those by pure prussic acid, the principal difference being that in the former they are perhaps less instantaneous or immediate in their accession, and that the duration of life is somewhat longer. Nevertheless, cases of very rapid death from this oil are recorded.

Dr. Taylor mentions the particulars of a case referred to him, in which it was probable that the whole duration of the case did not exceed seven minutes, and the man was not seized by the peculiar symptoms of poisoning until five minutes after he had taken the dose. During this time he was conscious and self-possessed, and replied rationally to questions put to him.

In a case related by Mertzdorff, in which 2 drams (7.75 gm.) of ethereal oil of bitter almonds were swallowed, death occurred in half an hour. Another one, by Dr. Taylor, is remarkable not only for its termination in a similar, short space of time, but from the fact of the smallness of the dose, which was only 17 drops. A druggist swallowed ½ ounce of "almond flavor," equivalent, it is said, to 30 drops of the oil. He fell insensible in less than half a minute. This case presents, moreover, this peculiarity, that there was a temporary remission of the symptoms. He was sensible for a few minutes, and spoke on the nature of his attack, but gradually again relapsed into a delirious and, apparently, very happy state. His eyes were extremely brilliant, but the pulse was quick and intermittent, and the whole body cold. He gradually recovered from the effects of the poison.[84] This case is a very extraordinary one; the peculiar effects cannot well be attributed to the smallness of the dose, since, as we have already seen, little more than half the quantity has proved fatal, and in this case it was strong enough to produce almost immediate insensibility. Mr. Iliff has reported a case in which death must have been very rapid. It is that of a young woman who poisoned herself in the Zoological Gardens, London. A small vial, containing a drop or two of the oil of almonds was found in the pocket of her dress, with the cork pushed in.[85]

[83] Lancet, June 8, 1844.

[84] Lancet, Sept. 1839, p. 930; Mr. Chavasse.

[85] Lancet, April, 1850.

In addition to the similarity in symptoms, the post-mortem appearances from this substance resemble those of prussic acid, as well as those of other narcotics. The same placid and natural expression of countenance, and the same purplish color and fluid condition of the blood, are here found. But the odor is uniformly present, generally more or less about the mouth, but, in all the cases yet reported, very characteristic, and penetrating into the stomach and cavities of the body generally. It is very persistent, and may be discovered several days after death.

There is a case related by De Keyser, which appears to show that the application of this oil to the skin may be dangerous. A lady used about half an ounce of it by mistake for an oil intended to make her hair grow. That the vapors of the oil did not occasion the symptoms is possible, because the waiting maid, who assisted her, was unaffected. She was seized with coldness, extending from the head and spine to every part of the body, followed by ringing in the ears and unsteadiness, deafness, swimming of objects before the eyes, and impaired power of moving the limbs. She fell insensible and in a state of collapse resembling syncope. She gradually regained her consciousness, but the sense of coldness continued for several hours.[86] It is not improbable that she may have inhaled the vapor.

431. Fatal use of apricot kernels.—These, also, may yield hydrocyanic acid. At Arles, a child ate two or three apricots; but, not content with this, also took the kernels inclosed in the fruit. Very soon after he was seized with convulsions, and died in spite of every attention.[87]

432. Fatal use of peach kernels.—Dr. Keating, of Philadelphia, has reported a very interesting case, in which he succeeded, by affusion of cold water, in restoring a child three years of age, who had eaten a quantity of peach kernels. The child was seized suddenly, and, when seen, was found insensible, with slow, deep, sobbing respiration, no convulsion of the limbs, but slight twitching of the mouth, icy-cold extremities, finger nails livid, hands slightly clenched, eyes prominent, and pupils dilated. A strong odor of prussic acid was perceived about the mouth. An emetic brought up a quantity of peach kernels, emitting the characteristic odor. It should be here observed that in these cases the hydrocyanic acid would be reasonably slow in being disengaged from the kernels in the stomach, and thus

[86] Jour. f. Pharmakodyn, 1857, p. 588.
[87] Quoted in the Am. Jour. of Med. Sci. Jan. 1853.

postpone the severe symptoms which arise from the ingestion of the acid itself. Another case is reported, in which the kernels of the cherry proved fatal to a child of five years, after forty hours' illness.

433. Amygdalin.—Amygdalin is contained in bitter almonds, cherry pips, peach, almond, prune, and apricot kernels; when these are swallowed, the emulsin contained in these kernels decomposes amygdalin, with the aid of the gastric juice, into hydrocyanic acid; this is accomplished slowly. According to Liebig and Wöhler, 17 grams of amygdalin yield 1 gram of prussic acid and 8 of oil of bitter almonds; 34 parts of amygdalin, mixed with 66 parts of almond emulsin, give a fluid equaling in strength that of the dilute hydrocyanic acid (U. S. P.) *viz.*, 2 per cent; theoretically, 100 parts of amygdalin will yield nearly 6 parts of hydrocyanic acid.

434. Fatal use of cherry-laurel water.—The following case of poisoning by this liquid is remarkable for the slowness with which the symptoms supervened, and the unusually long duration of life. A hypochondriac of advanced age drank, one morning, 1½ ounces (46 c. c.) of cherry-laurel water. The symptoms of poisoning did not come on for three hours. Then the hands and feet became paralyzed, and the head fell forward upon the chest. Involuntary discharges from the rectum and bladder took place. The extremities, though cold and immovable, were not insensible. The pulse was small, the voice hoarse but distinct, and the intellect perfect. The patient observed with pleasure the incessant progress of the weakness; he died in the evening without pain or convulsions. On section, the blood was found gluey, and of a peculiar, dark color; but no odor of bitter almonds was detected. The celebrated trial of Capt. Donellan, in 1781,[88] on a charge of poisoning Sir Theodosius Boughton with this liquid, is no doubt familiar to the reader. It is not definitely known how much hydrocyanic acid is contained in cherry-laurel water, since it varies with each preparation. It is known, however, that in summer, the distillate from cherry-laurel leaves contains almost twice the amount of acid as that distilled in winter, the amount gradually increasing from winter to summer. The leaves do not evolve the hydrocyanic acid, unless broken or bruised. Cherry-laurel water also becomes weaker by being kept. The medicinal dose is from 40 minims to 1 fluidram (3.89 c. c.).

435. Poisonous action on animals.—The effect of poisonous action on animals varies only from that on man, by the different method of respiration as between the cold-blooded and warm-blooded animals,

[88] Taylor, op. cit. p. 593.

and in the method of absorption due to the different anatomical arrangement for food digestion; for instance, warm-blooded animals breathe oxygen through the lungs, cold-blooded animals (as frogs) receive oxygen through their cutaneous coverings, as well as by the air passages; consequently, absorption of the acid solution in frogs is rather difficult, but they are susceptible to the action of the cyanid salts.

436. Post-mortem appearances.—It should be admitted that the peculiarities of these, in prussic acid poisoning, are not well marked, and that the general condition of the body is that usually met with after death from an intense narcotic, or death from suffocation or asphyxia. The face is either livid or pale; the lips and nails blue; and the skin of the neck, back, and shoulders much discolored. The jaws are firmly closed; the muscles of the hands and feet contracted; and cadaveric rigidity comes on sooner, and is more perfect than usual. The eyes have a peculiar brilliant and glistening appearance, the pupils are widely dilated, and foam is sometimes seen about the mouth. Evidence of involuntary evacuation of the bladder and rectum is not unfrequently observed. The veins of the brain are found turgid, and the lungs are congested with a very dark-colored but liquid blood. Orfila says that the mucous membrane of the air passages has generally a dark red color, which cannot be removed by washing, and the bronchial tubes are filled to their extremities with a bloody froth. More recent authorities would hardly admit this description by Orfila, since experience has shown that this poison usually destroys life by asphyxia, or by paralysis of the heart's action (see *supra,* § 422), and the post-mortem appearances from hydrocyanic or prussic acid are such as are met with, where the death is to be attributed to either of these causes.

The heart is usually found with the right side relaxed and engorged with liquid blood (having ceased its motion during diastole rather than during systole). This is the usual concomitant of death by asphyxia, and presents nothing unusual. The mucous membrane of the stomach is, in perhaps the majority of cases, highly reddened, and this deepening of color may extend for some distance into the intestinal tube. In a case reported by Jochner, and in a few others, a chocolate-colored fluid was found in the stomach. The blood is generally dark and fluid, sometimes of a purplish color. It will readily be seen how insignificant are the pathological alterations found in those who have been killed by prussic acid. There is no one of the appearances here noted which may not be met with in

death from many other causes, and especially in sudden death by some mode of asphyxia. The peculiar mottled and ecchymotic appearance of the lung tissue, the engorgement of both ventricles and sometimes of both auricles with dark fluid blood, the congestion of vessels of brain and spinal cord (Tardieu), mentioned by some authorities, are probably due to the asphyxia which is produced in the majority of cases of intoxication by this drug. Certain experiments,[89] performed by the writer, would seem to prove, not only this point, but also that rapid and early post-mortem muscular rigidity would follow the majority of deaths which suddenly occur to persons previously in full health and muscular activity. In some of these experiments upon the intoxication of animals by prussic acid, artificial respiration prevented convulsions and certain post-mortem appearances usually attributed to this poison. Probably this exception to the post-mortem appearances of asphyxia may be explained on the ground that the artificial respiration has prevented asphyxia, but the poison has caused its fatal effects from the interference with the functions of the nerve centers by preventing tissue oxidation.

In addition to the above post-mortem appearances of the congestion of the venous system generally throughout the organs and tissues of the body, there is usually a bloody foam in the bronchial tubes, and the lung tissue is often edematous in portions; ecchymoses are observed in the pleura and other serous membranes. Especially when the autopsy is made immediately after death, the odor of hydrocyanic acid is apparent; but it is not persistent, and does not continue as long as when death has been produced by nitrobenzol, the latter odor persisting for a longer period than in the case of hydrocyanic acid poisoning.

Kobert,[90] in his table of post-mortem findings, mentions the "post-mortem spots of markedly bright red color." Ecchymotic spots—especially of the lung substance—are usually seen in death caused by asphyxia. It should be remarked that these spots of a bright red color might possibly differentiate between death by asphyxia or drowning, as distinct from hydrocyanic acid asphyxia. Certain authorities mention the appearance of bright red or violet patches,[91] in the dependent parts of the body and of the finger nails,[92] as probably due to the formation of cyanmethemoglobin.[93] It should not be

[89] *Vide* London Practitioner, April, 1872, p. 197. Also a paper by the writer, with experiments, Boston Med. and Surg. Jour. Aug. 23, and Sept. 13, 1866.

[90] Op. cit. p. 33.

[91] Tichter, Prag. med. Wochenschr. 1894, Vol. XX. p. 105.

[92] Taylor, Brit. Med. Jour. 1892, Vol. II. p. 1168.

[93] Kobert, Lehrbuch der Intoxikationen, 1893. p. 515.

forgotten that we have already spoken of the cherry-red color of the blood, and its fluidity, as a characteristic post-mortem evidence of death by inhalation of carbon monoxid which also prevents tissue oxidation.

In formulating a differential diagnosis as to the cause of death by either of these poisons, the spectroscope may determine the fact, as the absorption bands of the two are in a different position in the spectrum. In regard to the ecchymotic spots, sometimes mentioned as found in the stomach mucous membrane, it should be observed that this is more likely to occur, where the death has been caused by a poisonous cyanid salt, and is not so likely to occur from the administration of the hydrocyanic acid itself. It should be especially noticed that the odor of hydrocyanic acid may be found in the contents of the skull, when first opened, and after it has disappeared from the body.

437. **Potassium cyanid and other poisonous cyanids in general.**—Potassium cyanid is taken as an example of all of the poisonous cyanid salts. These are sodic and ammonic cyanids, mercuric cyanid, silver cyanid, hydric sulphocyanid, potassium sulphocyanid, cyanogen cyanid, methyl cyanid, cyanuric acid. At the present time, toxicologists are of the opinion that the action of these cyanid salts is due to the decomposition in the stomach by means of an acid (gastric or hydrochloric), resulting in the splitting up of the salt into its base and the acid with which it was combined; consequently, the poisonous action of the salt would be shown by the action of the hydrocyanic acid, called "cyan poisoning." The local action upon the stomach would result from the irritating action of the base with which the acid was combined to make a salt, set free from its combination with hydrocyanic acid. Hydric cyanid, formerly called anhydrous hydrocyanic acid, of course would exhibit the same symptoms with more energy than hydrocyanic acid itself, and the local action upon the stomach and viscera would be irritant, showing an aggravated reddening of the mucosa, as well as ecchymosis and injection of the blood vessels.

We have already, in the previous pages, mentioned death following the ingestion of a solution of potassic ferrocyanid with tartaric acid, from which cyanid poisoning occurred. A similar action takes place if the ferrocyanids are mixed with a solution of peptone and casein at the same temperature as in the animal body, 37° to 40° C., (97° to 100° F.); but in this case the evolution of the cyanic acid is very slowly performed, and the amount is so small that at no given mo-

ment is injury likely to follow the ingestion in the stomach; and yet there might be a condition where the evolution would proceed more rapidly. It would also be noted that Volz[93a] reports the death of a man who swallowed a ferrocyanid salt of potash, and, after it, equal parts of nitric and hydrochloric acids; his death occurred within the hour, and with symptoms of cyanid poisoning. It is now generally admitted that the other cyanid salts, above named, produce death as the result of cyan poisoning.

Cyanid of potassium, as probably all salts of hydrocyanic acid, is equally destructive and almost as rapid in effects with the free hydrocyanic acid. The symptoms are exactly similar to those produced by the latter poison, as are also some of the post-mortem appearances. Dr. Finnell reports having met with intense redness of the gastric mucous membrane in three cases of fatal poisoning by this salt.[94] But Dr. Schauenstein found a dark red color of the membrane with bloody points, in only two out of five cases,[95] and attributes this peculiarity to the caustic operation of the solution in a concentrated state, and when the stomach contains but little food. The odor of prussic acid is less striking, and less frequently perceived in poisoning with this salt. In a case of sudden death from it, reported by Casper, there was no unusual odor, although the nature of the poison was detected by chemical analysis.[96] The reaction of the contents of the stomach is always alkaline, and, according to Schauenstein, prussic acid can always be detected in them by the addition of formic acid. The reason of this is that the cyanid of potassium is a very unstable salt, so that it is easily decomposed by weak acids; even the carbon dioxid in the air is sufficiently strong, hence the odor of hydrocyanic acid evolved from a bottle containing cyanid of potassium. This explains, also, why, in some cases, we may have the effect of a caustic, since a large portion of the cyanid may have been converted into carbonate of potassium, thus liberating the hydrocyanic acid. The quantity capable of proving fatal may be stated at from 2½ to 5 grains (0.162 to 0.324 gm.) of the cyanid salt, since the former quantity is equal to 1 grain (0.065 gm.) of anhydrous prussic acid. The fatal dose must necessarily vary with the strength of the preparation, and this is very different for different specimens. The strongest is made by saturating a solution of potassa with prussic acid.

A man, aged thirty, died in a quarter of an hour after taking 15 gr. (1 gm.), prescribed for him by his medical attendant by mistake

[93a] Blyth, Poisons, etc., op. cit. p. 210.
[94] Am. Med. Times, I. 33.
[95] Prager Vierteljahr. LXV. Anal. 14.
[96] Vierteljahrsschrift, July, 1854.

for the ferrocyanid.[97] Dr. Perry related the following case to the Boston Society for Medical Improvement: A nurse administered this poison by mistake to a child, who had a slight cough, instead of a cough mixture, which stood near the bottle of solution of the cyanid. The immediate effects of the dose were vomiting and convulsions; then insensibility, locked jaw, coldness of extremities, which were pendulous and without muscular power; diminished frequency of respiration (12 to 16 per minute), the pulse small but distinct, 60 per minute; the circulation languid, pupils dilated, sphincters paralyzed; the teeth closed so firmly and continuously that only once or twice could anything be poured into the mouth. Dr. P. saw the child in fifteen minutes after the accident, and found it in a warm bath, and insensible. It was treated by stimulants and the inhalations of ammonia. Death was sudden, and no post-mortem examination was made. The child lived one hour and a half.

A case is reported by Dr. C. E. Ware, of a woman who died in less than an hour from taking 7 grains (0.45 gm.) of this salt in a teaspoonful of liquid. Death occurred by gradual syncope.[98] In Vienna, Dr. Schauenstein met with five cases of fatal poisoning by this substance, in the course of eighteen months. In all of them the death seems to have been sudden. In one case, in a young girl, strong tetanic spasms came on directly after the poison had been taken.[99] The same symptoms, with severe abdominal pains, occurred in a case reported by Prof. Wagner, of Leipzig.[100] The root and the juice of cassava (Jatropha Manihot) produce symptoms identical with those of prussic acid, but, in general, they are less intense. De Keyser relates that three children who had eaten of the former, and an adult negro who had drunk about 6 ounces of the latter, recovered.[1]

438. Lethal dose of potassium cyanid.—The proportion of hydrocyanic acid contained in its cyanid salt of potash is 10 to 20 per cent, and, consequently, a dose of 2½ to 5 grains (0.16 to 0.32 gm.) would, theoretically, be its lethal dose. Practically the minimum fatal dose of potassium cyanid is about 2.4 grains (0.154 gm.). Recovery has followed, frequently, from much larger doses; but it is known that the strength of the commercial preparations is variable, and we therefore refer only to the doses of the pure salt. Three cases are quoted by Peterson and Haines as recovery after "what was esti-

[97] Henke's Zeitsch. Bd. 45 H. I. p. 6.
[98] Boston Med. and Surg. Jour. Dec. 1856. p. 387.
[99] Brit. and For. Med.-Chir. Rev. Oct. 1859, p. 530.
[100] Archiv. f. phys. Heil. 1859. p. 417.
[1] Jour. f. Pharmakodyn. 1857, p. 586.

mated to be at least 20,[1a] 40,[1b] or 60 grains[1c] (1.2 to 3.84 gm.) of potassium cyanid. In most of these cases the exact strength of the preparation was not determined."

439. Tests in general.—A portion of the solution to be tested is made alkaline with potassium hydrate, and then added to a mixture of ferrous sulphate and ferric chlorid, and gently warmed. Then the whole is acidified with hydrochloric acid. If the amount of hydrocyanic acid is large, a precipitate of Prussian blue will appear at once, and the solution is blue; in the presence of small amounts of hydrocyanic acid, however, the liquid is colored green, and the precipitate of Prussian blue appears only on standing for some time. Wormley maintains this reaction is sensitive to 1 part in 25,000.

A portion of the solution is rendered alkaline with sodium or potassium hydrate, and then a few drops of yellow ammonium sulphid are added, the whole evaporated to dryness, taken up with water, and a few drops of an acidulated solution of ferric chlorid added. If the original solution contained hydrocyanic acid, the addition of the ferric chlorid will give a red color, due to ferric sulphocyanate. According to Link and Möckel this is the most delicate test for HCN, and serves to detect 1 part in 4,000,000. Wormley states it is of about the same delicacy as the previous test.

A little of the solution is treated with a solution of silver nitrate, and acidified with nitric acid. On boiling, the silver cyanid is dissolved; while silver chlorid, if present, is not. The silver cyanid is also soluble in ammonia.

To a portion of the solution is added a dilute solution of potassium nitrite, then a few drops of ferric chlorid solution and enough dilute sulphuric acid to produce a yellow color. Warm the mixture, cool, and precipitate the iron with ammonia. To the filtrate add a few drops ammonium sulphid, when a violet color, passing through blue and green into yellow, is produced. Vortman places the delicacy of this test as 1:312,000.[1d]

A piece of filter paper, dipped in a 5 per cent tincture of guaiacum, and dried, then moistened with 0.1 per cent $CuSO_4$ solution, turns blue in the presence of HCN. Fallacies: Ozone, ammonia, chlorin, bromin, and other substances.

If compound cyanids are present, the material, instead of being distilled in the presence of tartaric acid, is thoroughly mixed with

1a Higgins, Med. Record, 1891, Vol. XL. p. 687.
1b Ord, Lancet, 1886, Vol. II. p. 1174.
1c Gillebrand, *ibid.* 1876, Vol. II. p. 223.
1d Monatschr. für Chem. II. 416.

sodium bicarbonate, and then distilled slowly. The cyanids are decomposed by the carbonic dioxid evolved, and the hydrocyanic acid is found in the distillate.

If mercury is found present, the mercury cyanid is decomposed by sulphureted hydrogen before beginning distillation, and the use of bicarbonate of sodium is determined by the presence of the compound cyanids.

440. **Test from odor of hydrocyanic acid.**—The only circumstance which is at all deserving of attention, and which merits a separate consideration, is the presence or absence of the odor of prussic acid. It may be at once stated that, where this odor is unequivocally detected, the evidence is satisfactory, since it is of so peculiar a character as not to be readily mistaken for anything else, unless nitrobenzol or oil of bitter almonds be excepted (see *infra*). Unfortunately, however, it is not always discovered, even in well-attested cases of poisoning by this substance. The odor is sometimes observed about the mouth and nostrils of the deceased, and is not perceived in the stomach. This was the case, in an instance reported by Jochner, of a young man who committed suicide by this poison. On the other hand, the stomach may exhale the odor of prussic acid, and none be perceived about the mouth or in the room. This was noted in the case reported by Mr. Hicks, in which, moreover, the examination of the body was not made until ninety hours after death. On opening the chest, the odor was more plainly perceived than in any other part of the body, and the fluid contained in the stomach smelled very strongly of prussic acid.[2] In none of the epileptic patients before mentioned was the odor of prussic acid discovered in any part of the body. The inspection was made twenty-four hours after death. Dr. Christison's case of recovery from a large dose may be referred to here, as corroborative of these facts; the first liquid, drawn from the stomach by the tube which he introduced, gave indications of the presence of prussic acid, on analysis, but not by any peculiar odor, although there was none other by which it might have been concealed. The stomach of Sarah Hart, poisoned by Tawell, had no odor of prussic acid, yet 1 gr. (.065 gm.) anhydrous acid was obtained by distillation from its contents, consisting partly of apple pulp. In the greater number of cases, however, there can be no doubt that it is readily distinguishable, since in some it has been so strong as to seriously affect the bystanders. The circumstances which cause these singular variations have not been thoroughly investigated. It is supposed,

[2] Med. Gazette, XXXVI. 460.

very naturally, that the length of time the person has survived after taking the poison, and the interval elapsing between death and the inspection of the body, must, as well as the dose, have an influence upon the preservation of the odor. But it is evident that these conjectures are not entirely satisfactory, since not only has the odor been detected after as long an interval as seven days, but, on the other hand, it has not been detected even where the presence of the acid has been demonstrated by chemical analysis. It is probable that in these cases it may have been fixed by a base. It is a matter of some importance to refer to a case (see § 7, *ante*), mentioned before, to show in what way a person with criminal intent can mislead justice by inhalation of some other odor more pungent than that of prussic acid, just before death. This, of course, might mislead the senses, but would in no way interfere with the chemical research for the poison.

441. Detection after death.—Notwithstanding the readiness with which prussic acid undergoes decomposition, it has been detected in the body after death. Rieckher detected it in the contents of the stomach, by the sulphur test, twenty-four hours after death.[3]

Mr. West was able to detect it on distillation, by the odor, and the silver and iron tests, twenty-three days after death, although no pains had been taken to insure its preservation.[4] In the following case it will be seen that it was detected after about the same period of time.

A young man of Tours having purposely poisoned himself with medicinal hydrocyanic acid of the twelfth degree, of which he appeared to have swallowed about 25 grams, M. Brame was called upon, after the lapse of three weeks, for the purpose of trying whether it was possible to detect hydrocyanic acid in the body. He was able to detect and estimate a considerable quantity of this poison which had remained in the stomach. By the addition of neutral and pure nitrate of silver, there was formed an abundance of a flocculent and yellowish precipitate, which, well washed, and dried under the air pump, and then heated for a few seconds on a sand bath, assumed a gray color. This precipitate was soluble in ammonia and cyanid of potassium. Treated while hot, with potassium hydrate, cyanid of potassium was formed, with which it was easy to obtain hydrocyanic acid and Prussian blue. Suspended in water, and subjected to the action of a current of hydrosulphuric acid, it gave rise to a clear and limpid solution of sulphocyanic acid, when the sulphuret formed had

[3] Canstatt's Jahresbericht für 1852, Bd. VII. p. 49.

[4] Prov. Med. Jour. July 23, 1845.

been separated by filtration. By means of hydrochloric acid, hydrocyanic acid could be obtained from it, of a very powerful odor, and the vapor of which formed a white precipitate in a solution of nitrate of silver; the precipitate was soluble in ammonia. The first precipitate, heated in a lamp, in a narrow tube closed at one end, gave hydrocyanic acid and a few drops of water, etc. This same precipitate, gently heated with caustic potassa, gave rise to no disengagement of ammonia.

In this case the hydrocyanic acid had remained in the stomach three weeks after burial. It did not appear to have entered into any chemical combination. There was a very considerable quantity of it, for, says M. Brame, "I was able to collect about 0.60 of cyanid of silver, or nearly 0.120 of hydrocyanic acid."[5] In the "Eaglesham" poisoning case it was detected in the stomach of the body, which had been buried fourteen days;[6] and in a German case, three weeks after death.[7]

442. Isolation.—It is advisable to try some of the preceding tests, (§ 439) Nos. 1, 2, 3, and 5, with the vapor of the material, before the distilling process about to be described. This can be done by inverting a watch glass, containing a drop or two of the proper solution, over the mouth of the jar containing the material to be examined, allowing the drop to dry, and then proceeding with the proper test.

Before proceeding with the distilling process for isolating hydrocyanic acid, it is necessary to test for potassium ferrocyanid, ferricyanid, and sulphocyanate, since these substances may form HCN on distillation with acids.

If mercury and compound cyanids are found absent, resort is made to the following process:

This process consists in placing a portion of the suspected material, mixed with a sufficient amount of water, if necessary, to render it fluid, in a retort which is connected with a condenser, the tube of which ends in a flask or receiver. If the fluid is not acid, a sufficient amount of tartaric acid should be added to give it a decided acid reaction. The retort is then heated, care being taken not to allow the temperature to rise over 105° C. or 110° C. As soon as two or three cubic centimeters of the fluid have collected in the receiver, the latter should be removed and another one put in its place. In this first distillate, if prussic acid were present in the suspected substance,

[5] The Chemist, Feb. 1855, from Comptes Rendus, No. 20, Nov. 13, 1854, by M. Brame.

[6] Edin. Med. Jour. IV. 163.

[7] Brit. and For. Med.-Chir. Rev. April, 1860, p. 531.

the odor can usually be perceived, even when it could not be detected in the original substance, and in some cases where the vapor tests could not be obtained; with this distillate the above tests should be employed both by the vapor and the liquid method. The distillation should be continued, until the distillate gives no further turbidity with a solution of nitrate of silver, and during the process the receivers should be changed as soon as a few cubic centimeters have been collected.

For the quantitative estimation of hydrocyanic acid, a solution of silver nitrate, slightly acidified with nitric acid, is added to the distillate in slight excess, the silver cyanid collected on a filter, washed, dried, and weighed. If the precipitate is not perfectly white, it should be treated with ammonium hydrate, which will dissolve the cyanid, leaving the sulphid on the filter. The latter is then dried, and weighed, and the previous weight of silver cyanid corrected. From the ammoniacal solution, the cyanid of silver is recovered by acidifying slightly with nitric acid, filtering and washing the precipitate. This precipitate, suspended in water in a flask, is then decomposed with as little hydrochloric acid as necessary, and the silver chlorid precipitate, after shaking the contents of the flask, allowed to settle. The supernatant liquid is filtered and subjected to the above tests.

VI. Oxalic acid.

443. Occurrence.—Most of the cases of accidental oxalic acid poisoning have resulted from mistaking the acid for Epsom salts, as the general appearance of the two is very similar.

It is widely distributed in nature, being formed as a salt in many plants, such as rhubarb, cinchona, nightshade, and sorrel. It is also a constituent of normal urine, generally in the form of the calcium salt.

Oxalic acid is largely used in dyeing and calico printing, and in bleaching processes,—especially of straw,—and in the household, as a bleaching agent for ink spots and cleaning woodwork.

444. Properties.—Oxalic acid, $(COOH)_2 + 2H_2O$, is generally made by oxidizing sugar or starch with nitric acid, or by the action of fused alkaline hydrate upon sawdust. It is a white, colorless body, crystallizing in prisms, or sometimes in needles, soluble in water, but more freely so in alcohol, with a very acid taste, and acid re-

action to blue litmus. On exposure to the air, it effloresces slightly, losing its two molecules of water.

445. Potassium binoxalate.—Potassium binoxalate is a salt commonly used in the household, under the name of "salts of sorrel" or "essential salts of lemon," as a bleaching agent for straw hats, or the removal of ink stains. It has been mistaken for cream of tartar, which it resembles in appearance and taste. Its effect upon the animal economy is similar to that of oxalic acid, and its detection is practically the same.

446. Statistics of poisoning from oxalic acid.—During five years (1883–87), in England and Wales, there were reported by the registrar general 120 cases of death by this poison. During twenty-one years (1870–91) there were 14 deaths from accidental poisoning by oxalic acid in New York city. During the twenty-five years, as shown in our table (pp. 18, 20) there occurred, in Massachusetts, four deaths by this same poison, 3 of which were accidental, and 1 from suicide. Witthaus and Becker found, in the current medical literature prior to 1895, 83 deaths from suicide, and 41 from accidental ingestion. Mr. Blyth reports 223 deaths—200 from suicide—as occurring in ten years (1883–1892) in England and Wales. Apparently, it is not a common poison used to commit murder, which may be understood when its forbidding, sour taste is recognized. Christison, as early as in 1836, calls attention to this well-recognized acid flavor of oxalic acid, and characterizes its rapid and fatal action.

In this treatise on poisons (1836) is the following statement: "If a person, immediately after swallowing a solution of a crystalline salt which tasted purely and strongly acid, is attacked with burning in the throat, then with burning in the stomach, vomiting, —particularly of bloody matter,—imperceptible pulse, and excessive languor, and dies in half an hour, or still more, in twenty, fifteen, or ten minutes, I do not know any fallacy which can interfere with the conclusion that oxalic acid was the cause of death. No parallel disease begins so abruptly and terminates so soon; and no other crystalline poison has the same effect." It is well to remark, as opposed to this statement, that Taylor[7a] reports the shortest interval of fatal poisoning, as the cause of death by arsenic, to be twenty minutes; this poison, indeed, has not the extremely sourish taste possessed by a salt of oxalic acid, and, therefore, Christison's statement must be taken with all its conditions.

[7a] On Poisons, 1875.

447. Preparations.—The only officinal preparation (U. S. P.) used as a medicine in this country, containing compound of oxalic acid, is oxalate of cerium, which is not usually admitted as a poisonous drug. Acid oxalate of potassium (potassium binoxalate, KHC_2O_4 H_2O) is commonly in use under the name of "essential salt of lemon" or 'salt of sorrel," and should always bear a label "poison," because it is commonly used to remove rust and ink stains from white cloth, and for scouring brass and copper. In its amorphous form, it resembles, both in appearance and taste, bitartrate of potash, "cream of tartar." This oxalate salt possesses all the poisonous action of the acid itself.

448. Symptoms of poisoning.—After the hot and sour taste in the mouth, vomiting is usually the first symptom, and is attended with burning pain and constriction in the throat and stomach, although it does not always occur, and in some cases the pain is absent. The vomited matters are dark-colored, and contain blood and sometimes membranous shreds, and highly acid, greenish, dark-colored, or bloody mucus. Abdominal pains are also present, but oftentimes the symptoms of nervous prostration and collapse with stupor and lack of sensation follow so rapidly the ingestion of the poison that the gastric disturbance is not a very prominent symptom, and the pain is not always observed. If, however, the symptoms of collapse are not present early in the case, the sickness of the stomach and vomiting are very distressing, the abdominal pain is severe, the extremities are benumbed and drawn up, the surface is cold, and the pulse irregular or imperceptible. A degree of stupor or drowsiness is sometimes observed. The urgency of the symptoms depends upon the degree of dilution in which the salt is taken; for, in the case of weak solutions, the local irritation upon the mouth, throat, and stomach, is less marked, and the symptoms of nervous prostration predominate, though they may come on more slowly.

In a case quoted by Dr. Christison, no vomiting occurred for seven hours, except when emetics were administered. The person had taken ½ ounce (15.55 gm.) dissolved in 10 parts of water, and diluted immediately afterwards with copious draughts of water. In another case, a man swallowed one ounce, and immediately drank warm water; he was not seen until fourteen hours after he had taken the poison, and in the mean time had traveled a distance of ten miles.[8] When, on the other hand, the acid is given in a highly concentrated form, the irritant and corrosive action upon the mouth, throat, and

[8] Lancet, Sept. 1845, p. 293.

stomach, becomes the most prominent symptom, and may be immediately followed by collapse, stupor, complete paralysis, and death. In this latter case the symptoms are not dissimilar to those of any other irritant or corrosive.

The rapidity with which a fatal result ensues varies a great deal. In some cases the action of the poison is extremely rapid. Mr. Iliff reports a case in which death appears to have been immediate. The wife of a chemist drank a saturated solution of oxalic acid in her husband's shop; she was found dead by the side of the counter, where she had probably fallen and died without a struggle.[9]

Dr. Christison quotes a case in which a young man survived ten minutes; another, in which a woman, who swallowed 2 ounces (62 gm.) died in twenty minutes; and Dr. Taylor refers to a case where death ensued in three minutes. The quantity taken in the last case could not be ascertained. Pereira also mentions a case which he examined, in which a man died in twenty minutes after swallowing oxalic acid in mistake for Epsom salts.

449. Subacute poisoning.—Although death usually occurs within a few hours, cases are mentioned in which it was postponed for several days. If the acute symptoms, produced by the local action of this poison, do not, on account of the concentrated dose, destroy life, oxalic acid or its potassium salt, when taken in a diluted solution, may be then absorbed. In these cases, which may also occur if the dose is small, the action of the drug is exerted upon the nerve centers.

450. Chronic symptoms.—Mr. Blyth remarks that, "with both oxalic and the acid oxalate of potash, certain nervous and other sequelæ are more or less constant, always time being given for their development."

Dr. Jackson reports one case in which the poison did not prove fatal until the tenth day;[10] and in a case described by Dr. Frazer, death occurred from its secondary effects upon the twenty-third day. Some instances of recovery are reported. An interesting one was observed by Dr. Didama, in which a woman dissolved two large tablespoonfuls of oxalic acid, by mistake for Epsom salts, in a small quantity of water, and took it on an empty stomach. Some twenty minutes afterwards she vomited, at first the solution she had taken, and then a dark-colored, bloody-looking fluid, in which were numerous white membranous patches. Ipecacuanha, and afterwards pre-

[9] Lancet, Oct. 1845.
[10] Boston Med. and Surg. Jour. Vol. XXXI p. 17

pared chalk, were administered to her, and in about an hour she was found quiet and nearly free from the intense, burning pain in her stomach and throat. She subsequently vomited again, and discharged from her bowels a large amount of matter resembling that which she had vomited. She soon recovered entirely, and returned to her work. A similar case, in which 1 ounce (about 31 gm.) was taken, and the patient recovered in eighteen days, is reported in the Lancet;[11] and a few others are referred to by Dr. Taylor.

Action.—The action of oxalic acid, except in the rapid and fatal cases of acute poisoning, has not been finally explained. It undoubtedly produces a disturbance of the respiration, and enfeebles the action of the heart, and makes its contractions irregular (arrhythmic); it also causes nervous symptoms, as shown by spasms and muscular languor; sometimes, cerebral disturbances would suggest that narcotic intoxication occurred as a consequence of a prolongation of the acute symptoms, but paresis has been observed in some of the lower animals, though not in man. Dogs are not as easily poisoned as man. This has been explained on the supposition that the presence of lime in the alimentary tract causes a combination of oxalate of lime which is comparatively an inert drug; yet, if the doses are pushed far enough, this animal will succumb to the usual poisonous action of oxalic acid. Orfila records a case in which there was marked slowing of the pulse and soporific tendency.

So long ago as in 1823, Christison[12] published his opinion that, in addition to direct action of oxalic acid as a corrosive poison to the primæ viæ, this acid, as well as its potassium and sodium salts, were violent poisons. From the experiments on the lower animals by Kobert and Küssner,[13] solid oxalate in toxic doses will at first paralyze the respiratory system, and death then supervenes from asphyxia; while with nontoxic doses there is no change from normal, respiratory movements, and no alteration of the normal pulse. If the dose be repeated in small amount, and frequently, the symptoms of subacute and chronic poisoning are observed, the respiration becomes slower and slower, and then ceases by the gradual paralysis of the respiratory muscles. The first symptom of poisoning, observed in both subacute and chronic forms, was a soporific condition and the reflex action weakened. The muscular action of the hind legs was also weak, and gradually deepened into a complete paralysis. This paralytic condition gradually extended into the fore legs. On the

[11] July 1846, p. 39.
[12] Edin. Med. and Surg. Jour. 1823.
[13] Exper. Wirkungen der Oxalsäure, Virchow's Archiv. Bd. LXXVII. s. 209, also *ibid.* Bd. LXXXIII.

sixth day the hind legs lost the reflex action and were motionless. Küssner and Kobert noted that, in all their experiments, lethal doses of these oxalates, given to animals, developed the appearance of sugar in the urine.

Christison mentions, in his treatise on poisons, a case in which healthy leeches died when applied to a patient who had taken a large dose of oxalic acid: "They fastened immediately; on looking at them a few minutes afterwards, I remarked that they did not seem to fill, and on touching one it felt hard, and instantly fell off, motionless and dead; the others were in the same state. They had all bitten, and the marks were conspicuous; but they had drawn scarcely any blood. They were applied about six hours after the acid had been taken." This would seem to show that oxalic acid is absorbed into the blood.

Two cases are mentioned by Taylor, in one of which 120 grains (about 8 gm.) were swallowed; there appeared a weakness in the sound of the voice; in one of these the normal strong and deep tone became, in nine hours, low and feeble, and continued in this condition for a month; in addition, he complained of a numbness and tingling of his legs.

451. Lethal dose.—Though a dose of 60 grains (3.88 gm.) is known to have caused the death of a boy sixteen years of age, yet the usual fatal dose for an adult is generally admitted to be four times this amount; and this opinion is supported by statistics of mortality. Recovery has occurred after a dose of 2 ounces (about 63 gm.).[14]

The smallest quantity which has proved fatal is recorded by Dr. Barker (as above mentioned), in which case it was roughly estimated that about a dram (.4 gm.) was eaten "as a dry solid" by a boy of sixteen years, who was found an hour afterwards in an insensible, pulseless condition with the jaws spasmodically closed. He had vomited some bloody mucus; his tongue and lips, though not injured by the local contact of the acid, were unusually pale. He died within nine hours from the time of taking the poison.

Generally speaking, it is very difficult to obtain a definite knowledge of how much has been taken, since this substance is usually, as before remarked, mistaken for Epsom salts or common salt, and, not being as agreeable to the taste as either of these salts, the whole quantity taken out is not generally swallowed, but thrown away. So, again, the concentration of the solution

[14] Lancet, Dec. 1, 1855, and Taylor on Poisons, Phila. 1875.

of the poison may increase the danger from the initial symptoms; and, in the case of a diluted solution, medical skill may combat the more tardy appearance of the general constitutional symptoms, and thus avert a fatal catastrophe from a comparatively large dose. It, however, by no means follows that a smaller quantity might not be attended with a fatal result, since some persons appear to have been far more seriously affected than others by equal amounts of this poison. This is proved by the above case of a lad, sixteen years of age, who ate 60 gr. (3.4 gm.) of dry oxalic acid, and died in nine hours. The only manner in which the quantity capable of destroying life can be approximately ascertained is by reference to such cases as have proved fatal, in the absence of medical assistance. The binoxalate of potassium, salt of sorrel, or essential salt of lemons, produces the same symptoms as oxalic acid, and destroys life as readily. An instance is reported by Chevallier, in which death took place in ten minutes.[15] In another case, a third dose of 1½ drams (5.83 gm.) proved fatal in an hour.[16]

452. Duration of symptoms.—The shortest period recorded[17] of death from this poison is reported by Ogilvie; the immediate cause was due to a hemorrhage of the stomach, and death was without pain, in three minutes. In acute poisoning, death usually occurs from the local action on the mucous membrane within twenty-four hours; in subacute poisoning within three to five days; and in chronic poisoning within a month.

453. Treatment.—As in the treatment of poisons, the local action of which is corrosive, the use of the stomach pump is unadvisable, on account of injury to the tissues of the primæ viæ. By referring to the case of Dr. Didama, recited above, it will be observed that the correct treatment for the immediate corrosive action would be the administration of prepared chalk and an unirritating emetic. It should be remembered that the fixed alkalies (sodium and potassium) form, with oxalic acid, an oxalate which is soluble, while magnesium oxalate and calcium oxalate are insoluble. The two former soluble salts are poisonous. Calcined magnesia or its carbonate should be preferred, or even the wall plaster of a room.

454. Post-mortem appearances.—It is stated that death may ensue from oxalic acid, and yet no traces of its action on the alimentary canal be observable on section; this occurred in a case where an ounce had been taken,[18] and a similar case has been seen by the

[15] Ann d'Hygiène, 1850, Vol. I.
[16] *Ibid.* 1842.
[17] Lancet, 1845, Vol. II. p. 205.
[18] London Med. Repos. Vol. III. p. 380.

writer. This is certainly not the ordinary result. According to Dr. Geoghegan, who examined the stomachs of three persons poisoned with this acid, the mucous membrane of the stomach was softened, with various shades of brown discoloration, and erosion or gelatinization: there was a brownish-black, ramiform vascularity of the submucous tissue, and the contents of the stomach were of the color of coffee grounds, owing to the action of the acid upon the mucus, and coloring matter of the blood. In Dr. Jackson's case, small ulcerations and thickening of the mucous membrane were observed, together with permanent redness. Hence, the action of the acid may be chiefly expended upon the mucous coat of the stomach, no actual corrosion being observed. In an instance reported by Mr. Letheby, the coats of the stomach were so softened that it could scarcely be handled without lacerating it, and in another, referred to by Dr. Christison, it is said that this organ was perforated.[19] In some of these cases, however, it is evident that the condition spoken of may have resulted from the long contact of the acid with the coats of the stomach after death, since even so feeble an acid as that naturally contained in the stomach is capable of producing similar results. It is certainly not the ordinary effect of oxalic acid. The œsophagus is also, in many cases, found altered, having a scalded or boiled appearance. It is pale, brittle, corrugated, and abraded in some places.

Fifty years ago, a case was reported by Dr. A. Wood, in which the stomach presented a large, irregular aperture. As sufficient details of the section are not given, and the viscera do not appear to have been examined *in situ*, it is possible that this hole may have been artificially produced. The autopsy was made thirty-five hours after death.[20]

Schmaus and Ewing present, in their work on pathology to which we have previously referred, the following post-mortem appearances in acute poisoning by oxalic acid: "Oxalic acid and its potassium salt produce white or grayish eschars, which may be yellow from imbibition of hematin, or brown or yellow from bile. The gastric mucosa is not corroded, but transparent, edematous, hyperemic, or bleeding; its contents are brownish. White, opaque deposits on the mucosa of the stomach and intestines are due to amorphous or crystalline oxalate of lime, and this is found also in the renal tubes. Post-mortem perforation may occur. The vessels contain black thrombi."

[19] Med. Gazette, Vol. XXXV. p. 49. Lond. 109; Wood's Materia Medica and Med. Repos. Vol. XI. p. 20; Toxicology p. 199, Phila. 1882.

[20] Edin. Monthly Jour. March, 1852.

Küssner and Kobert found[21] that, in animals who died after the subacute symptoms appeared, the kidneys were pale from a diminished amount of blood; and that a peculiar stripe was marked, conforming to the canaliculi in the cortical substance, which endured for several weeks. This showed, under the microscopical inspection, that it had been caused by the deposit of crystals of oxalate of lime; this was confirmed by chemical tests, which proved the presence of a large amount of oxalates. Oxalic acid would not be likely to remain in the tissues free, because the presence of lime in these tissues would form a base of oxalate of lime.

Kobert and Küssner[22] report that the urine contains, during life, a strong reducing agent of unknown nature, calcic oxalate crystals in abundance, and usually albumin and renal tube casts.

There is also a certain condition of the system called oxaluria, which is associated with certain diseases attended with impaired oxidation, in which traces of calcic oxalate may be recovered from the urine; but the insignificant amount, as compared with that in which a poisonous dose of oxalic acid has been taken, would settle any doubt, if such existed. This doubt, however, shows the importance of making an estimate of the amount of oxalic acid which should be attributed to a case of poisoning.

A case of suspected poisoning by this acid was reported[23] in detail by Dr. J. C. White, of Boston, to the Society of Medical Sciences. The post-mortem appearances were as follows: Twenty-four hours after death the stomach, intestines, spleen, kidneys, liver, and part of the pancreas were removed from the body and brought to Dr. White for examination, and forty-eight hours after death examination of the viscera showed their condition as follows:

The stomach, which, with 6 inches of œsophagus attached, was tied at both extremities, contained 10 or 12 ounces of grayish fluid of the consistence of gruel, with an acid smell and reaction. It was marked externally by dark streaks, corresponding to the position of the blood vessels, and by large stains of a scorched appearance near the œsophagus. Within, its lining membrane, for an inch or more surrounding the œsophageal opening, was of the same black color as that tube, and the blood vessels, in the depending portions filled with black blood, were visible through it. Elsewhere, and generally, the

[21] Op. cit. Pathology and Pathological Anatomy, p. 362.

[22] Exper. Wirkungen der Oxalsäure, Virchow's Archiv. Bd. LXXVII. s. 209, also *ibid.* Bd. LXXXIII.

[23] Boston Med. and Surg. Jour. Jan. 27, 1870, p. 57.

mucous membrane was of its natural color or paler than usual. The structure of the mucous membrane and other tissues was unchanged, except in the lower portions, where it was soft and thin (probably a cadaveric change). There was more or less mucus attached to its inner surface.

The œsophagus, exteriorly, had a uniform, deep slate color. Its interior surface was stained a deep black, the same color penetrating in parts to and through the muscular tissue. The lining membrane was not materially softened nor easily separated from the tissues beneath.

In the intestinal canal there was nothing unnatural besides the usual cadaveric softening of the mucous membrane, and the reddened patches.

All the other organs were apparently healthy, the blood contained within them being generally bright red, or vermilion.

Christison states that, after concentrated doses, the "stomach is found to contain black extravasated blood, exactly like blood acted upon by oxalic acid out of the body."

Medical Examiner Draper returned (Case 280, 1902) to the Massachusetts secretary of state the following notes of an autopsy of a death from poisoning from oxalic acid: Countenance distressed. Lividity of dependent parts. No evidence of poisoning about lips or tongue. Heart normal. Lungs injected. Stomach contained 6 ounces of turbid fluid. Liver and kidneys injected. Spleen and pancreas normal. Brain congested. Note: Chemist found oxalic acid.

455. **Detection and tests.**—Unless antidotes have been given, the stomach contents or vomited matters will have a very strong acid reaction. If antidotes have been given, the reaction may be neutral or even alkaline.

Oxalic acid may be found in two conditions, either as the free acid, or as a salt combined with calcium, magnesium, or organic matter.

If oxalic acid or oxalates are the only things to be tested for, the following is the best method of separation:

The material, if strongly acid, is to be diluted with water and then strongly agitated, so that all the oxalic acid will be in aqueous solution. It is then filtered, the residue being saved for the examination for insoluble oxalates. The filtrate is then evaporated to a thin syrup, and then a large excess of alcohol added, which will coagulate any dissolved proteid, and at the same time leave the oxalic acid in

solution. This alcoholic extract is then filtered (save the residue for soluble oxalates), and the filtrate evaporated on the water bath till all the alcohol is removed, and the residue taken up in a little water. This solution will then contain any free oxalic acid that may have been present in the original mixture. The residue precipitated by the alcohol (proteids, etc.) is next acidulated with hydrochloric acid and extracted with alcohol, filtered, the filtrate evaporated and taken up with water, which will then give a solution of oxalic acid that may have existed as a soluble oxalate, originally. The first residue insoluble in water is next to be treated with enough potassium carbonate to render it alkaline, and then boiled for an hour or two. This will decompose any insoluble oxalates into potassium oxalate. The filtrate is then evaporated to a small volume, and made acid with hydrochloric acid, and extracted with alcohol. The alcoholic filtrate is evaporated, and the residue taken up with water, when any oxalic acid that may have existed as an insoluble oxalate, in the material, at the beginning, will be found in solution.

Another method for the separation of oxalic acid from organic mixtures may be employed as follows:

Liquids containing organic matter, as in matters vomited or in the contents of the stomach, should, if very acid, be diluted with water if necessary, strained, and filtered; the filtrate will contain all of the oxalic acid which existed in the free state in the liquid. This oxalic acid should then be precipitated by a solution of acetate of lead; the resulting oxalate of lead collected upon a filter paper, washed, then suspended in water, and a current of sulphureted hydrogen gas passed through the mixture until there is no longer any oxalate of lead undecomposed. The fluid will then contain a black precipitate of sulphid of lead, and oxalic acid and sulphureted hydrogen in solution; filter, and evaporate upon a water bath; when a residue will be left, which is more or less pure oxalic acid, and which can then be examined by the appropriate tests. If, however, antidotes have been administered, such as chalk or magnesia, the supernatant liquid, after standing some time, may, if not acid, be thrown away, and the residue treated with dilute hydrochloric acid, which will dissolve the oxalate; filter, wash, and treat the filtrate with a solution of acetate of lead as above described. The precipitate will then contain chlorid of lead mixed with the oxalate, but this will not interfere with the isolation of the oxalic acid by the above method, since the subsequent evaporation will volatilize the hydrochloric acid, as well as the sulphureted hydrogen.

With a solution of oxalic acid, soluble salts of calcium, such as the acetate or chlorid, yield a white precipitate of calcium oxalate, insoluble in ammonia or acetic acid, soluble in strong hydrochloric or nitric acid. If the precipitation is done very slowly, it will often come down crystalline, which is easily recognizable.

Oxalic acid, in solution, gives, with silver nitrate solution, a white precipitate of silver oxalate, soluble in ammonia or nitric acid (difference from hydrochloric acid). If this precipitate is collected on a filter, washed, dried, and heated upon platinum foil, it explodes. Lead acetate gives, with oxalic acid, a white precipitate of lead oxalate, soluble in nitric acid, insoluble in acetic acid. Oxalic acid heated on platinum foil sublimes completely. Potassium binoxalate does not sublime, but is converted into potassium carbonate, alkaline to moist red litmus paper, and effervesces with a drop of hydrochloric acid.

VII. Picric acid.

456. Properties.—Picric acid, $C_6H_2(NO_2)_3OH$, trinitrophenol, is a yellow, crystalline body, slightly soluble in water, freely soluble in alcohol. It possesses strong coloring properties.

457. Occurrence.—The use of picric acid in the arts as a dye, also in the manufacture of lyddite, emmensite, and melenite, also for coloring foods and beer, has caused accidental poisoning; one case, where it was used to produce suicide, is recorded[24] by Karplus. The workmen employed in factories have absorbed this poison through the skin. The local action is irritant both to the cutaneous and the mucous surface.

458. Symptoms of poisoning.—Its internal ingestion is followed by pain in the abdominal region, vomiting, and diarrhea; the urine shows a yellow color, which, on exposure to the air, becomes a reddish-brown. There may be suppression of urine, or difficult micturition with pain. The yellow color may appear in the vomited matters and in the fecal discharges; the skin, and the mucous covering of the eye, may also be stained yellow. An itching eruption occurs, sometimes, on the hands of those who work with it in factories. The absorption of this poison is accompanied with prostration, stupor, and sometimes causes convulsions and collapse. The elimination of the poison with the urine is slow, and the yellow stain of the skin may persist for several days.

[24] Zeitschr. f. Klin. Med. 1893, Vol. XXII. p. 210.

459. Subacute and chronic poisoning.—This is principally observed among factory hands, and the symptoms are irritation of the mucous surface of mouth, nose, stomach, and the intestinal tract. An application of picric acid to the vagina has turned its mucous membrane yellow, and the effects of its absorption have been shown by a consequent attack of gastrointestinal disturbance, red urine, somnolence, and distress in the stomach.[25]

460. Action.—Like many other inorganic poisons,—especially of the nitro combinations,—picric acid has the effect, after absorption, of combining with the hemoglobin of the blood corpuscles, and of displacing the weaker combination in them of oxyhemoglobin, thus preventing the normal metabolism of the tissues. This combination is known as methemoglobin, and can be recognized by its peculiar absorption band in the spectrum.

461. Treatment.—Removal to a pure, warm air, cleaning the skin of any stain of the acid, emptying the contents of the stomach, and, to combat the failing strength of the circulation, the administration of such medicines as will stimulate the heart's action; but alcoholic stimulation is contraindicated.

462. Detection and tests.—Thoroughly extract the material with strong alcohol. Evaporate off the alcohol, adding water, if necessary, to a syrupy consistency; and, after making acid with sulphuric acid, extract with ether, using, if necessary, successive portions of the latter, and acidifying each time. Unite the ethereal extracts, add a little water, and evaporate all the ether. With this aqueous solution the following tests may be made:—

A woolen thread immersed in a solution of picric acid (colored yellow), rinsed lightly in water, and the color extracted with ammonia, is colored red by solution of KCN. Cotton thread is not stained the preliminary yellow. An alkaline solution of a picrate, on warming with concentrated solution of KCN, forms potassium isopurpurate which imparts a red color to the liquid. Glucose and an alkali also impart a red color.[26]

VIII. Tartaric acid.

463. In general.—One case in which this substance incontestably proved poisonous is related in the Lancet, January 2, 1845. A man took, by mistake for Epsom salts, an ounce of tartaric acid dissolved

[25] Cheron Jour. de Thérap. 1880, Vol. VII. p. 132.

[26] Zeitschr. f. anal. Chem. IV. 185.

in ½ pint of warm water. He immediately exclaimed that he was poisoned, and complained of a violent, burning pain in his throat and stomach. Obstinate vomiting continued for nine days, when he died. Nearly the whole of the alimentary canal was found highly inflamed. Tartaric acid was detected in the dregs of the cup, and the druggist admitted that he had made a mistake. Another case is reported by Devergie, but the accuracy of his opinion and analysis is contested by Orfila.[27] Christison mentions an instance in which 6 drams of tartaric acid were taken in twenty-four hours, without the least inconvenience, and both he and Coindet administered it to cats without observing any injurious effect. An instance is on record in which the bitartrate of potassium proved fatal by excessive purging, but the quantity swallowed, or rather eaten, is said to have been more than a quarter of a pound.[28]

464. Symptoms of Poisoning.—The symptoms of poisoning by this irritant seem to result in consequence of the severity of vomiting and diarrhea induced by the local action upon the digestive tract, rather than from any specific mode of action of the agent. It would seem as if this article belonged to the class of substances, mentioned in our first chapter as producing serious symptoms from excessive or abnormal doses, which are not with ordinary use classified as poisons.

In Mr. Blyth's table (p. 15) there is recorded but one case of poisoning by tartaric acid during the ten years in England and Wales, and no deaths were returned by the medical examiners of Massachusetts.

Dr. Luff notes that this substance is not regarded commonly as a poison; yet he mentions that the above-recorded case was the subject of a trial before the Queen's Court for manslaughter.[28a] Trevithick records[29] a fatal case of poisoning, which is mentioned by Luff as having been self-administered by a woman aged sixty-seven. The woman confessed to having taken a dose of about 3 ounces in very strong solution. The ingestion was followed by severe pains in the belly, vomiting, and diarrhea. Feverish symptoms set in (100° F.), and, on the fourth day, delirium occurred with a failing pulse, which condition persisted until her death. The diarrhea, which had been checked by opium and bismuth, recommenced the day before her death.

465. Post-mortem appearances.—These appear as an acute, inflam-

[27] See Ann. d'Hygiène 1852, Vol. I. pp. 199, 382, and Vol. II. p. 320.

[28] Lond. Med. Gazette, 1837–38 I. 177.

[28a] Reg. v. Watkins. C. C. C. 1845. (Cited by Luff, op. cit. Vol. I. p. 377.)

[29] Brit. Med. Jour. 1893.

matory condition of the whole digestive tract, or, rather, of the mucous membrane which lines the gullet, the stomach, and the intestinal canal; this is particularly emphasized in the transverse and the descending portion of the colon. There may also be seen a few shallow erosions in the lining membrane of the gullet, and spots of hemorrhages in the submucous tissue of the stomach.

466. Detection.—The chemical detection of tartaric acid is very difficult, because it forms so large a constituent of ordinary food and of cooking salts (cream of tartar); therefore, in any case of poisoning by tartaric acid, it is necessary to isolate a large quantity from the stomach. Since tartaric acid is decomposed before being absorbed into the blood, it is, of course, useless to search for it in the blood or solid viscera. To isolate it, the contents of the stomach are diluted with distilled water if necessary, filtered, and the filtrate divided into two equal parts; one portion is exactly neutralized with potassium hydrate or carbonate, then added to the other portion, the mixture shaken actively and allowed to stand for some time, when nearly all of the tartaric acid will be precipitated in the form of crystalline cream of tartar (acid tartrate of potassium).

IX. Ethyl alcohol.

467. Occurrence.—Ethyl alcohol, CH_3CH_2OH, spiritus vini, is made from saccharine liquids by the growth of various varieties of Saccharomyces or yeast plants. In this fermentation 94 to 96 per cent of the sugar employed is converted into alcohol, the remaining 6 to 4 per cent being transformed into other products such as "fusel oil," succinic acid, glycerin, etc.

The various alcoholic beverages vary widely in the percentage of alcohol they contain. Those obtained by fermentation of malt liquors, such as beer, ale, porter, stout, contain from 1 to 9 per cent. Those obtained by fermentation of grape juice, such as light wines, contain less than 12 per cent; and heavy wines, port, sherry, Maderia, Marsala, contain from 16 to 25 per cent. Those obtained by distillation, called "spirits," such as brandy, whisky, rum, and gin, contain from 40 to 60 per cent. Fortified wines are, as a rule, heavy wines which have been "fortified" by the direct addition of alcohol. "Fusel oil" is a mixture of various higher alcohols the chief of which is primary amyl alcohol, or isobutyl-carbinol.

468. Properties.—Pure ethyl alcohol, absolute alcohol, is a colorless, mobile, volatile liquid, with a characteristic odor and burning

taste (sp. gr. 0.8095 at 0°C., 0.7939 at 15°C.; b. p. 78.4°). Solidifies at 130.5°C. It burns with a nonluminous flame. It is hygroscopic, and is miscible with water in all proportions, giving out heat, and contracting in volume. It is neutral to litmus paper.

469. **Poisonous nature of alcohol.**—The pernicious effects upon the system, of the abuse of alcoholic liquors, are too well known to need any mention here. We propose, therefore, to refer only to their immediate poisonous action, when taken in large quantity into the stomach.

Death, from this rapid saturation of the system with alcohol, is by no means rare. Orfila mentions an instance in which a man died immediately from the effects of a large dose of brandy.[30] Dr. Rösch relates three cases in which adults died from the immediate effects of excessive drinking, in a few hours.[31] Taylor says that a man died in half an hour after swallowing a bottle of gin for a wager. Rösch also relates the cases of two children in which quite a small quantity proved fatal. The one was a boy aged two years, who drank some brandy and soon after became comatose, had convulsions, and died in a few hours. The other was a little girl of four years, to whom her uncle had given about two tablespoonfuls of spirits. The child soon sank down insensible, was seized with convulsions, and, in spite of all remedies that were used, died within twenty-four hours. In another case, the same quantity of brandy was given to a child six months old to keep it quiet during the night. In less than a minute it was attacked with convulsions; its face was purple, the eyes staring, the pupils dilated and insensible, the mouth open, the head extremely hot, while the rest of the body was cool, the breathing stertorous, and the pulse hardly perceptible. It had also bloody evacuations. It died, in a state of coma, in nine hours.[32]

In general, the state of stupor is preceded by a short period of great excitement; but in some cases this preliminary stage is either very short or entirely absent. The difference probably depends upon the strength and quantity of the spirit, and the age of the person.

Mention should be made here of the foolish and criminal bets that have caused sudden and frightful deaths from drinking a large quantity of some alcoholic beverage. This has been observed, not only among those who have, by long experience, acquired a certain immunity from the use of excessive doses of alcohol, but

[30] Op. cit. II. 528.
[31] Henke's Zeitschrift, 1850, 4 H.
[32] Deutsch Canstatt's Jahresbericht für 1851, Bd. 10, p. 286.

also among persons little used to alcoholic excesses, and who have allowed themselves to drink a quart of alcohol, hardly stopping a moment to take breath. These unfortunates fall, as if struck down, into a profound coma, accompanied by little convulsive muscular shocks. Respiration, at first stertorous, becomes more and more difficult, and a bloody froth appears on their lips. Involuntary evacuations occur, and they die, sometimes in an hour or a half hour, and sometimes as soon as fifteen minutes after the fatal drink.

Since the former edition of this work the following additional cases of acute alcoholic poisoning have been returned by the medical examiners of Massachusetts.

1885. Half-witted laboring man, induced by bystanders to drink a large quantity of whisky, was found dead twelve hours later.

1890. Medical Examiner Brown (case 18) found a man insensible, who died in less than twelve hours after drinking alcoholic liquor.

1893. (Case 128) Medical Examiner Draper. Woman drank a dipperful of raw brandy and died thirteen and a half hours later.

1895. (Case 1) Medical Examiner Carleton. Child six years old found dead beside intoxicated mother. Second case, Medical Examiner Adams (case 18). Alcohol poisoning in child two years old.

1896. (Case 178) Medical Examiner Harris. Child twenty-six months old.

1898. Pint of whisky drank, on a wager, by a man who died in ten minutes after drinking.

1900. (Case 138) Medical Examiner Draper. Child eight months old drank enough whisky to kill him by convulsions and coma, in twelve hours.

1902. (Case 18) Medical Examiner Brown. Boy of fifteen stole alcoholic liquor from a wagon, found insensible, and died in less than twelve hours after drinking.

470. Symptoms of poisoning in general.—The general characteristics of the comatose stage in the adult are the following: The face may be either pallid or flushed; the pupil at first contracted, and afterwards dilated and insensible to light; respiration slow and sometimes stertorous; the pulse quick and jerking; and the limbs cool and relaxed. In general, the appearance is very much the same as in poisoning by opium, or as in the apoplectic condition. In the absence of any knowledge of the mode of accession of the symptoms, the diagnosis of the case will often be incomplete. The odor

of alcohol in the breath is, of course, an uncertain sign, since ardent spirits may have been swallowed, without being the cause of the symptoms. The ability to arouse the patient temporarily is also no means of distinction, as this may be possible in the stupor from intoxication, and opium poisoning.

In fact, should the individual die, the physician will often be left in doubt as to the cause of the symptoms until some evident explanation for them is found in the post-mortem examination. Even then nothing may be found to throw light upon the case, since a person presenting the above symptoms may have died of concussion of the brain, which leaves no ascertainable morbid change. If the symptoms have been due to opium or other narcotics, these may not be discovered, and, as will presently be seen, the evidences of death from alcohol may also be deceptive. It will only be from a careful analysis of the history of the case, and comparison of it with the post-mortem signs, and by isolating a considerable amount of alcohol from the tissues and viscera, that the physician can hope to come to a probable conclusion.

We quote from Kobert the following description of alcoholic intoxication, or acute poisoning: "Loss of control of the motor nerves of locomotion; staggering gait or inability to walk; absence of sensation; reddening of the face; stertorous breathing; vomiting; involuntary stools; pulse hardly noticeable; sticky skin, cyanotic; temperature and blood pressure greatly reduced (Katzenjammer)."

471. **Paper on acute alcoholic poisoning read before Massachusetts Medico-Legal Society.**—The following extract, in fact the whole paper on acute alcohol poisoning, was read before the Massachusetts Medico-Legal Society at its meeting in June, 1880;[33] and, since it covers the ground so completely, from a medico-legal point of view, no apology is required for its introduction here.

I. Acute Alcoholic Poisoning.

"The pathological anatomist meets with two classes of cases of alcohol poisoning, the acute and the chronic. The former is not unfrequently the immediate cause of death, the latter rarely so. That fatal cases from acute poisoning are not so rare as might be supposed is shown by the fact, mentioned by Taylor, that in four years (1863–1867) thirty-five deaths from this source occurred in England and Wales alone.[34]

[33] Medico-Legal Relations of Alcoholism, its Pathological Aspects, by G. K. Sabine, M. D., Med. Examiner for Norfolk Co. Mass.

[34] H. C. Wood, Therapeutics, Materia Medica, and Toxicology.

"In describing the post-mortem appearances it will be necessary to describe those belonging to the two classes separately. The statements of different observers vary greatly in regard to these appearances, and in many respects are entirely contradictory. This is due, no doubt, to the want of making a proper distinction between the acute and the chronic forms, many symptoms thus being considered as due to acute poisoning which are in reality met with in chronic cases only.

"Within a short time the effects of the different kinds of alcohol, in poisonous doses upon dogs, have been thoroughly investigated by Dujardin-Beaumetz and Audigé. These investigations were carried on by means of more than two hundred and fifty experiments upon as many different animals, and the general conclusions drawn from them were as follows:—

" 'In animals which succumb to acute alcoholic poisoning, anatomical lesions are constantly found, which vary in intensity with the strength of the alcohol. These lesions occur especially in (1) the digestive organs; (2) the circulatory and respiratory organs; (3) the nervous system; and (4) the kidneys.

" '(1) Lesions of the digestive organs.—In regard to the stomach the changes are less marked when the alcohol has been administered subcutaneously, and then only a slight redness towards the pyloric extremity is perceptible; but when the alcohol is administered by the œsophagus, the lesion is more marked, and, in certain cases, the mucous membrane is more or less softened. The effects depend on the strength of the alcohol, being much more marked when the alcohol is concentrated, and therefore more caustic.

" 'The alteration in the small intestine is more constant and more marked when the alcohol has been administered hypodermically. It exists more especially in the upper part of the intestine; but when death is protracted, it occurs throughout its whole length. The intestinal mucous membrane is then softened, its surface of a dark red color, and it presents, in the majority of cases, more or less abundant hemorrhages. These appearances may be accounted for by the elimination of the alcohol by the intestinal glands.

" 'In the large intestine a hemorrhagic appearance is met with,—especially towards its inferior extremity and on the longitudinal bands.

" 'The liver is the gland most seriously affected in acute alcoholic poisoning; this organ, always very quickly congested, is soft throughout and friable; it can be torn between the fingers, and the

hepatic cells may be destroyed in a great measure. The spleen is also engorged with blood, and its tissue softened. Finally, in a certain number of cases, the head of the pancreas is found to share in the congestion presented by the duodenum.

"'(2) Lesions of the circulatory and respiratory organs.—In acute alcoholic poisoning, the blood is essentially altered; it is blackish, and forms more or less abundant clots in the heart. The pulmonary lesions are characterized by a distention of the vascular system. This congestion is frequently more marked when the alcohol has been introduced by the stomach, and in these cases there are ecchymoses at the base of the lungs.

"'(3) Lesions of the nervous system.—Lesions of the cerebro-spinal axis are characterized by considerable venous congestion of the meninges. In the brain, the veins and sinuses are engorged with blood, and the gray matter is also somewhat congested. These cerebral lesions are more pronounced the longer the comatose period continues.

"'(4) Lesions of the kidneys.—These lesions are but slightly marked when caused by fermented alcohol, but, when due to œnanthylic or caprylic alcohol, are more pronounced. But it is, above all, in acute glycerin poisoning that they attain their greatest intensity; there exists then, not only a congestion of the organs, but a certain quantity of blood is found in the bladder.'

"Lallemand, Perrin, and Duroy (Alcohol et des Anesthésiques) state that 'marked anatomical changes are found in animals which have died from the administration of alcohol. The mucous membrane of the stomach and of the upper part of the small intestine is inflamed, sometimes quite violently. The liver is much congested. The lungs show no signs of asphyxia or even real congestion. The right side of the heart and the large veins are filled with very liquid, dark blood. The meninges are engorged with blood. The alcohol is diffused through all the tissues and secretions; but the brain, cord, and liver contain a much larger proportion than any of the other tissues.'

"So much for poisoning in the lower animals.

II. Alcohol Poisoning in Man.

"On inspection, the dependent portions of the body are found discolored by hypostatic congestion. It has been claimed by some that decomposition sets in very slowly, but this is denied by others.

"'The general appearances resemble more or less closely those of asphyxia; the right side of the heart, the pulmonary arteries, and the systemic veins being loaded with blood, while the left cavities and the arterial system are comparatively empty, the blood which they do contain being dark.[35] The sinuses and the whole venous system of the brain are turgid with dark blood.'[36]

"The blood is very fluid or imperfectly coagulated, and of a dark color.[37]

"The mucous membrane of the stomach is usually found very much injected, as indicated by either a bright[38] or deep red[39] color, covered with coagulated mucus (albumin),[40] and sometimes ecchymosed.[41] The congestion usually extends both into the œsophagus and into the small intestine.[42]

"The action of strong alcoholic liquid on the mucous membrane of the stomach so closely resembles the effects produced by arsenic and other irritants, as easily to give rise to the suspicion of mineral irritant poisoning.[43]

"The amount of alteration in the gastric mucous membrane will vary, of course, with the quantity of alcohol taken, its degree of concentration, and the amount of food, etc., in the stomach.

"A very few cases have been reported in which the mucous coat has been found in a sloughing condition. According to Baer, frequently no lesions are perceptible. In such cases the fluid has been taken very much diluted.

"Alcohol is found to be present in the contents of the stomach, in variable quantity, or entirely absent[44] from the same, according to the quantity ingested, the rapidity of absorption, and the time which has elapsed between the last potations and the death of the individual.

"The length of time that portions of it may remain in the stomach is consequently very uncertain. Taylor mentions a case in which a pint of spirits was taken, and a fatal effect produced in

[35] Carpenter on Alcohol.

[36] Woodman and Tidy, Forensic Medicine and Toxicology; Carpenter on Alcohol.

[37] Casper, Forensic Medicine, Vol. II.

[38] Taylor on Poisons; Christison on Poisons; Birch-Hirschfeld, Lehrbuch der Pathologischen Anatomie.

[39] Wood, Therapeutics, Materia Medica, and Toxicology; John J. Reese, Manual of Toxicology; Oesterlen, Heilmittellehre; Taylor on Poisons; Woodman and Tidy, Forensic Medicine; Birch-Hirschfeld, Lehrbuch der Pathologischen Anatomie.

[40] Oesterlen, Heilmittellehre.

[41] Wood, Therapeutics, Materia Medica, and Toxicology; Oesterlen, Heilmittellehre; Birch-Hirschfeld, Lehrbuch der Pathologischen Anatomie.

[42] Woodman and Tidy, Forensic Medicine.

[43] Taylor on Poisons.

[44] Oesterlen, Heilmittellehre.

eight hours; but no traces of it could be detected in the stomach. That it may entirely disappear from the stomach, long before it is eliminated from the other organs, is proved by a number of cases of more or less chronic poisoning related by Magnan. In one of these, three days and six hours had been passed without excess in drink; the liver and brain contained alcohol, but none was found in the blood or other organs. In another patient who had died fifty hours after his last potation, alcohol was found in the liver, brain, and blood, in very appreciable quantity; the lung also contained traces.

"When present, as it probably almost always is,[45] at the time of death, in cases of acute poisoning, the contents of the stomach will, of course, possess the odor of the liquor ingested, unless masked by some other substance present.

"The mucous membrane of the respiratory tract exhibits a widely spread and intense injection of the blood vessels;[46] the bronchi are filled with frothy mucus;[47] more or less hyperemia of the lungs may always be expected;[48] they are very often found in a state of edema; still more frequently hypostases and hepatization are discovered in their posterior and inferior portions.[49]

"The liver, kidneys, and spleen are usually found loaded with venous blood.[50]

"The presence of alcohol may be detected in the liver even after it has entirely disappeared from the stomach and all the other organs, with the exception of the brain; for it is these two organs which show the greatest affinity for it.[51]

"Nearly all authorities state that the veins of the cerebrum and cerebellum, together with their membranes, are engorged with blood.[52] According to Birch-Hirschfeld, however, the substance of the brain itself is frequently found anemic and edematous.

"According to some, an effusion of serum[53] is frequently found within the ventricles or beneath the arachnoid, and, according to many others, an effusion of blood[54] is very apt to take place,—espe-

[45] Christison on Poisons.

[46] Boehm, Ziemssen.

[47] Carpenter on Alcohol.

[48] John J. Reese, Manual of Toxicology; Casper's Forensic Medicine; Woodman and Tidy, Forensic Medicine and Toxicology; Wharton and Stillé's Medical Jurisprudence; Lallemand, Perrin, et Duroy, Alcohol et des Anesthésiques.

[49] Boehm, Ziemssen.

[50] Carpenter on Alcohol.

[51] John J. Reese, Manual of Toxicology.

[52] Oesterlen, Heilmittelehre; John J. Reese, Manual of Toxicology; Wharton & Stillé's Medical Jurisprudence, Pt. I.; Casper's Forensic Medicine; Taylor, Treatise on Poisons; Birch-Hirschfeld, Lehrbuch der Pathologischen Anatomie; Lallemand, Perrin, et Duroy, Alcohol et des Anesthésiques.

[53] Oesterlen, Heilmittellehre; Carpenter on Alcohol; Taylor on Poisons; Manual of Toxicology, John J. Reese.

[54] Tardieu, Etude sur l'Empoisonnement; Taylor on Poisons; Woodman and Tidy, Forensic Medicine and Toxicology; Christison, Treatise on Poisons.

cially in the latter locality. It is probably somewhat doubtful how frequently these effusions take place in the most acute cases. The statement that hemorrhagic apoplexy occurs quite constantly seems to be founded on a very limited number of cases.

"Through the kindness of Dr. Harris and Dr. Amory, of this society, I have been enabled to obtain the autopsy records of two cases of undoubted acute alcohol poisoning, which fell into their hands for examination; and Dr. Wood, of the Medical School, has kindly furnished me with an account of the result of the chemical analyses in the same cases. Dr. Draper has also published an account of the examination of a more or less chronic case, in the Transactions of this society, Vol. I., No. 1.

"Dr. Harris's case.—Two women of bad character were induced in the evening by sailors to go on board a vessel lying at one of the wharves, where they drank a large quantity of Spanish rum, known as aguardiente. At two in the morning one of the women was found dead.

"Autopsy fourteen hours after death. Rigor mortis marked. Lividity of dependent portions of the body. An abrasion of the mucous membrane in the lower part of the vagina, 1½ inches, was discovered. No marks of violence.

"Section.—The lungs were bound to chest walls—especially posteriorly—by not very firm adhesions. The heart presented no very unusual appearances except that the muscle was a little lighter colored than usual. The abdomen contained about one ounce of dark-colored fluid. The lungs were slightly congested, and the bronchi contained a little frothy mucus.

"The kidneys were firm, quite dark in color, with the pyramids very strongly marked. The internal surface of the stomach was intensely congested, the contents (about two ounces) smelling strongly of rum. The brain was of normal consistence, the vessels of the pia mater not overloaded; the puncta cruenta were, however, strongly marked. The uterus contained free, dark blood, and in the right ovary was a corpus luteum of menstruation. The intestines and bladder presented nothing unusual. The spleen presented appearances of old inflammation. The liver, brain, stomach and contents, were reserved for chemical analysis; the kidneys, part of heart, and uterus, for microscopical examination.

"Dr. Wood isolated from the contents of the stomach ½ c. c. of alcohol, 0.825 specific gravity, which contains 89 per cent of absolute alcohol, and from a half of the brain 2 c.c. of alcohol of the

same strength. This corresponds to four c.c. in the whole brain; and since 89 per cent is about double the strength of ordinary spirituous liquors, the amount found in the brain corresponds to about eight c.c. of ordinary liquor.

"Dr. Amory's case.—A man about forty-two years old left home in the company of a friend. Two hours later, after visiting a number of places where liquor was sold, and drinking freely, he was brought home in a comatose condition, and died shortly after.

"Autopsy seven hours after death. Body well developed. Face livid. A superficial cut just beneath left orbital ridge, from which a small quantity of blood was slowly oozing. Thick layer of fat under the skin. The deceased wore a truss for double inguinal hernia. Left inguinal canal open, the right closed. Diaphragm, on the right side opposite the fourth rib, and opposite the fifth on the left. Pigmentary deposits in both lungs. Old pleuritic adhesions on posterior surface of right lung, chiefly confined to the lower lobe. Pericardium contained about a half ounce of serum. Left ventricle contracted, right flaccid. Mitral valve admitted two fingers with difficulty, tricuspid three readily. No clots in heart or large vessels. The posterior portion of the lower lobe of right lung engorged with blood, but pressure caused crepitation. A small cyst about the size of a pea on the periphery of right kidney. The external surface of stomach considerably congested, the internal intensely reddened, showing the action of some irritant substance. The rugæ quite prominent. The contents consisted of fragments of meat, potatoes, and carrots, and about 1½ pints (by estimate) of fluid smelling of alcohol. No clot found in any part of body or blood vessels. Vessels of pia mater moderately filled with blood.

"Dr. Wood isolated from the contents of the stomach 33⅓ c.c. of 89 per cent alcohol, and about ½ c.c. of the same strength from one half of the brain.

"The medical examiner meets with another class of cases much more frequently than with the preceding. These are cases in which the subjects have been addicted for a long time, during life, to alcoholic excess, and have died during a debauch, the immediate cause of death being either some exposure to cold, some chronic disease caused by the long-continued use of alcohol, or the continued dose of alcohol in a system already very much weakened by its excessive use.

"In these cases one is apt to meet with certain appearances which belong to cases of acute poisoning, if alcohol has been recently

taken, such as redness of the gastric mucous membrane, fluidity of the blood, etc., and either few or many of the signs of chronic poisoning may also be present, but no one of them is constant or pathognomonic."

472. Symptoms of chronic poisoning.—Catarrhal formation of the mucous membrane of the mouth and primæ viæ; fatty degeneration of the liver, the liver first enlarged, then cirrhotic; blood vessels fatty and atheromatous; kidneys degenerated; decrease of visual faculties and of intelligence; muscular tremblings; boils on the skin; delirum tremens, and collapse (Quartal-saufen).

In both forms of acute and chronic alcoholic poisoning, and owing to a degenerated condition of the kidneys, the patient may have a sudden or slowly approaching case of pneumonia, from which he may die. Acute insanity, active or melancholic, may develop in a course of acute intoxication. The former condition may induce homicidal tendencies, the latter suicidal.

The symptoms of chronic alcoholism are more for the medical practitioner than for the toxicologist; but inasmuch as the post-mortem appearances may enshroud a doubtful case of suspected poisoning, it would seem advisable to present a careful *résumé* of the results of recent study, which is offered in the paper, herewith published by permission of its author, G. K. Sabine, M. D., medical examiner for Norfolk county, Massachusetts, upon—

"THE MEDICO-LEGAL RELATIONS OF CHRONIC ALCOHOLISM, AND ITS PATHOLOGICAL ASPECTS.[55]

"Although the acute form of alcoholic poisoning is not unfrequently the immediate cause of death, and the chronic alone rarely so, the two are so often combined that the signs of the latter should be familiar to the medical examiner.

"Of all the various conditions that are attributed to the habitual use of alcohol, but very few, if any, are pathognomonic. The statements of different observers vary greatly in regard to the post-mortem appearances of the chronic, as well as of the acute form, and the question to what degree certain diseases may be attributed to this form of poisoning remains an open one. In fact, almost every known chronic affection has been attributed some time or other to the intemperate use of alcohol. It is probably the fact that the re-

[55] Transactions of the Mass. Medico-Legal Society, Vol. I. No. 5, 1882.

mote is frequently looked upon as the immediate cause. That the habitual and long-continued use of alcohol so alters the tissues and impairs their functions that they are more prone to become diseased, there can be no doubt.

"Perhaps there is no question which the medical examiner is more frequently called upon to decide than whether or not he has to deal with a subject who has been an habitual drunkard.

"Although no single pathological condition is sufficient to determine this positively, yet there are certain ones which point strongly in this direction, and the same inference can be drawn from others, taken collectively, which are almost equally conclusive.

"Among the various pathological conditions resulting from chronic alcoholism are the following:—

"Changes in the skin.[56]—In the earlier stages of this affection the skin is remarkably smooth and soft, owing to an increase in the fatty tissue. According to Frerichs,[57] the secretion contains a larger amount of oil than normal, a condition similar to that which exists when cod-liver oil is taken in large doses for some length of time. Later on the skin becomes dry, and on the extremities hard and inelastic.

"Acne rosacea, consisting of an inflammation and even suppuration of the sebaceous glands, is among the characteristic symptoms of the intemperate use of alcohol. The nose and face are its favorite seat. Besides the nodules, the skin is reddened, owing to the dilated capillaries and consequent blood stasis, and is also infiltrated to a greater or less degree.

"The blood.—The most striking change in the blood is an increase in the watery elements and diminution in the fibrin. It contains much serum, forms no coagula, or only very small ones, and is of a very dark color; hence the term "venous plethora," used by some of the older authors. The dark color is explained by an increase in the hydrogen and carbon (Steinheim); or by a destruction of the red blood corpuscles (C. H. Schulz), so that less oxygen is taken up and less carbonic acid is given off. Another peculiarity presented by the blood is the increase of fat contained in it. It has been stated that not unfrequently the serum of blood, drawn from a person suffering from delirium tremens, is more or less opaque or even milky (Morgagni, Nasse), this appearance being due to an excessive amount of fat.

[56] The following statements are largely taken from Baer.

[57] Klinik der Leberkrankheiten, l. c. s. 319.

"Fatty tissue.—There is a marked increase in the subcutaneous fat, in the fat between the muscles, about the different organs, especially the heart, kidneys, intestine, in the greater and lesser omenta, in the mesentery, etc. In the later stages of alcoholism, when the digestion becomes impaired and the blood deteriorated, this accumulation of fat disappears. According to Rokitansky, there is an increase of fat in the marrow of the bones, the bony tissue, at the same time, being atrophied.

"In the earlier stages of the affection the increase of fat is due to an infiltration rather than to a degeneration. The fatty liver, for instance, is of essentially a different nature at this time from that met with later on in the disease.

"The stomach and intestine.—A chronic catarrhal condition of the stomach is quite constant, and appears early in the disease. This is indicated by abundant, soft, gray mucus, projections of the mucous membrane, and by the slaty color that occurs,—especially near the pylorus. Another form is very apt to be met with, which is characterized by circumscribed hypertrophies of the whole mucous membrane (*gastritis prolifera*), and produces little warty projections (*gastritis verrucosa*); later, larger polypoid growths result (*gastritis polyposa*).[58]

"Owing to the disturbance of circulation which takes place later in other organs, the return of blood from the stomach is interfered with, so that a varicose condition of some of the veins is produced.

"The hypertrophy is very apt to be accompanied by dilatation of the glands, due to compression at their outlet, so that small cysts, which are filled with a clear fluid and project from the surface, result. According to Klebs,[59] an inflammation of the submucous tissue may be produced by the excessive use of alcohol, and this go on to suppuration, or it may result in the formation of large masses of connective tissue, without destruction of the mucous membrane. In this manner it occurs at the pyloric extremity, producing stenosis.[60]

"The continued irritation of the diseased mucous membrane is productive of a variety of ulcerations, from the small, hemorrhagic erosion, characterized by a superficial loss of substance, to the so-called round or perforating ulcer.[61]

"According to Erbstein,[62] after administering large quantities of

[58] Orth, Diagnosis in Pathological Anatomy, p. 285.

[59] Handbuch der Pathologischen Anatomie, s. 179.

[60] Birch-Hirschfeld, Lehrbuch der Pathologischen Anatomie, s. 831.

[61] Klebs, Handbuch der Pathologischen Anatomie, s. 186.

[62] Virchow's Archiv. Bd. 55, s. 469.

dilute alcohol to dogs for three or four days, the peptic and ordinary gland cells of the stomach are found cloudy and granular. The lumen of the pyloric glands is plugged by a finely granular, yellowish, or yellowish-brown mass. In extreme cases, fat drops appear in the altered cells. In most cases, chronic alcoholism produces no marked effect on the intestine, although, in many, a chronic catarrh exists.

"The liver.—The liver is the first and the most severely affected by the abuse of alcohol, of all organs in the body. The alcohol, being taken up by the portal system, is carried directly to this organ, and there, by its irritating effect, produces various disorders according to the individual's condition, and more especially the character of the alcohol. The more concentrated the alcohol, the sooner and the more severely is the liver affected.[63] Beer and wine seldom affect the liver, and are almost never productive of severe forms of degeneration.[64] The most frequent affections of the liver produced by chronic alcoholism are simple fatty infiltration;[65] inflammation of the parenchyma, and fatty degeneration of the same;[66] and, lastly, inflammation and hyperplasia of the interstitial connective tissue.

"Among the causes of fatty liver, the abuse of alcohol is one of the most prominent. According to Frerichs it ranks only second, chronic disease of the lungs standing at the head of the list. He says: 'Of thirteen individuals who died of delirium tremens, in six the liver was very fatty; in three the organ contained little fat, and in two none at all; lastly, two died of cirrhosis of the liver.' It is probable, but not absolutely certain, that the alcohol acts by retarding the metamorphosis of tissue,[67] and the blood, being overcharged with fat, deposits it in this organ.[68]

"In higher grades the liver is enlarged, but usually appears flattened, the edges are generally thickened and rounded off. The peritoneal covering of the liver is transparent, smooth, and shining. According to the grade of fatty infiltration, the surface of the liver is yellowish-red or distinctly yellow. The consistence of the organ is diminished; it feels doughy, and pits on pressure with the finger. On incision we meet little resistance; a coating of fat remains on the warm knife blade. The cells in the periphery of the acini first

[63] Frerichs. A Clinical Treatise on Diseases of the Liver, Vol. II. p. 33. Sydenham Trans.

[64] *Ibid.*

[65] Frerichs. A Clinical Treatise on Diseases of the Liver. Vol. I. p. 229; Klebs, Handbuch der Pathologischen Anatomie, s. 382; Orth. Diagnosis in Pathological Anatomy, p. 318; Niemeyer. Text-book of Practical Medicine, Vol. I. p. 656.

[66] Klebs. Handbuch der Pathologischen Anatomie, s. 406.

[67] Text-book of Practical Medicine.

[68] Baer, Alcohol.

become infiltrated, and later on those nearer the center.[69] The quantity of blood in the capillaries is diminished in proportion to the amount of infiltration.[70] On microscopic examination, according to the grade of the disease, the enlarged or usually rounded liver cells appear filled with fine fat globules, or those have united to form single, larger drops, or, lastly, individual liver cells are entirely or mostly filled by one large drop of fat.[71]

"Fatty or granular degeneration of the liver is attributed to the abuse of alcohol by Baer, who quotes Klebs in so doing. The reason why the latter assigns this as a cause is perhaps owing to the fact that he makes a somewhat different classification of the diseases of the liver, considering certain forms as degeneration, which are looked upon by other authorities as simply infiltration. According to him, the direct action of other toxic substances upon the liver, such as hydrocyanic acid, carbonic oxid, phosphorus, arsenic, and antimony, produces this degeneration. Among the organic substances, alcohol, ether, and chloroform hold an important position.

"Interstitial hepatitis. Cirrhosis of the liver.—The most common cause of this form of interstitial hepatitis, which extends uniformly over the whole organ, is usually considered to be the intemperate use of alcohol, still this is not necessary; most drunkards do not have a cirrhotic, but a fatty, liver, and many persons with cirrhosis are not in the habit of dram drinking.[72] Certainly, cirrhosis is so commonly the result of the abuse of alcohol that, when met with, the cause may fairly be suspected.[73] Frerichs speaks of it as 'the chief cause.'

"Birch-Hirschfeld says that it is doubtful if cirrhosis is ever due to any other cause. The volume of the liver is increased or diminished according to the stage of the process. It is only accidentally met with in the early stages. The principal change produced in the liver consists in an increase of the interlobular tissue, and the appearance of small, grayish masses at the periphery of the lobules. The consistency of the liver is increased. The cause of this change consists of a growth of granulation tissue from Glisson's capsule, from which small projections extend into the acini. In the later stages of chronic interstitial inflammation the liver is more or less diminished in size, in rare cases fully a half; its surface is uneven

[69] Niemeyer, Text-book of Practical Medicine.

[70] Baer, Alcohol.

[71] Niemeyer, Text-book of Practical Medicine.

[72] Orth, Diagnosis in Pathological Anatomy; Frerichs, a Clinical Treatise on Diseases of the Liver; Niemeyer, Text-book of Practical Medicine.

[73] Baer, Alcohol.

and covered with prominences, which vary from a millet grain in size to that of a pea, and are usually of a yellow, icteric color. At the edge of the liver,—especially in front where it is sharp,—single nodules are frequently found, completely isolated, as the capsule belonging to the two surfaces comes in contact here. Upon section, a similar condition of things is seen in the interior of the organ.

"Organs of respiration.—Drunkards are very subject to catarrh of the larynx,[74] which is often accompanied by a similar condition of the pharnyx.[75] This catarrhal inflammation of the larynx not unfrequently extends into the bronchi.

"A very important question is whether the habitual use of alcohol predisposes to disease of the lungs. Upon this point authorities differ so widely that it is quite impossible to draw any conclusion.

"The heart.—In habitual drunkards the heart is almost always found hypertrophied. This hypertrophy may be brought about in various ways. As is well known, the effect of alcohol is to increase the frequency and force of the pulse. Whenever a muscle is called upon to do an extra amount of work, the effect is to increase the size of that muscle. This hypertrophy of the ventricular walls, which is simply the result of an increased amount of work, is also produced by various obstructions to the circulation which the heart has to overcome. Owing to the deposit of fat in and about the different organs, to the fatty infiltration of the cardiac muscle itself, the work of the heart is increased. This will also be caused by the disturbance of the pulmonary circulation, owing to bronchial catarrh, emphysema, etc., and also to disturbance of the portal circulation from fatty liver, cirrhosis, etc. Another very important factor in the cause of hypertrophy of the cardiac muscle, and especially of the left ventricle, is, according to some authorities, the condition of the kidneys frequently met with in chronic alcoholism. Here one may find fatty degeneration of the parenchyma, accompanied by an increase of interstitial connective tissue, which has become more or less shrunken. According to Traube this contraction cuts off a large number of small vessels, and results in an increase of pressure in the aortic system, producing dilatation and hypertrophy of the left ventricle. This explanation is objected to by others, for instance, Bamberger. Still another obstruction to the circulation is owing to the lumen of the vessels being increased; for when this occurs, the blood stream is rendered slower, and has to be overcome by increased

[74] Baer, Alcohol; Niemeyer, Text-book of Practical Medicine.

[75] Niemeyer, Text-book of Practical Medicine.

heart's action. Finally, an atheromatous condition of the arteries is to be mentioned, as this causes a decided obstruction.

"In the later stages of alcoholism a fatty degeneration of the cardiac muscle occurs, and, in the very last stages, owing to the general inanition at that time, the muscle becomes atrophied and diminished in weight. The organ is pale and flabby, diminished in size in all directions.

"The vessels.—The change in the capillaries consists in an increase in their lumen, that of the smaller and larger arteries in the so-called atheromatous degeneration. The dilatation of the small vessels, and passive hyperemia of all the organs have been explained on the ground that the alcohol has a paralyzing effect upon the vasomotor system; also that the alcohol, by its irritating effect upon the walls of the vessels, causes a fatty degeneration of the same, and, as a consequence, a loss of tonicity.

"Aside from the fatty condition, a sclerosis of the walls takes place, owing to hyperplasia; this may result in the so-called ossification, an infiltration of lime salts into the newly formed tissue. As a result of these conditions, the vessels lose their elasticity, become hard and stiff, and thus are more resistant to the flow of blood. This chronic inflammation of the walls of the vessels, which underlies this process, may be brought about by the continued use of alcohol. The irritation which alcohol produces in all the tissues may be sufficient to produce inflammation of the walls of the vessels. A still more important element in the causation is the constant stretching which the walls undergo, and which predisposes them to the atheromatous change. Traube attributes this degeneration in alcoholism to this, and also to the diminishing of the rapidity of the blood stream, which occurs at the same time. He says: 'The worst of these cases occur in drunkards. It is not unlikely that the increased tension of the aortic system which is observed in drunkards, not only while they are under the immediate influence of liquor, is owing to a contraction of the smaller arteries, which results in an increased tension, by interfering with the flow of blood from the aortic system. This being the case, the increased tension observed in the larger branches would be accompanied by a slowing of the blood stream.' The primary inflammatory condition may also be brought about by the overdistention of the smaller vessels, caused by the increased heart's action. When they have once lost their tonicity this increased action ceases. It is a well-established fact that, as the quality of the blood becomes deteriorated, a condition which is a constant accompaniment of the

general cachexia, the nutrition of the walls of the vessels is interfered with, and a fatty degeneration results.

"Affections of the urinary organs.—After each ingestion of alcohol, the secretion of urine is increased, as a larger quantity of water is excreted with it.

"The diseases of the kidneys which most frequently occur in drunkards, and especially in the latter stages of alcoholism, are the parenchymatous and interstitial or granular nephritis. This latter is divided into two stages, that of infiltration of cellular elements, and the other of connective-tissue formation. At first the inflammatory process produces an active hyperemia, with an exudation of fluid and white blood corpuscles into the interstitial connective tissue. This in turn is productive of anemia, impaired nutrition of the renal epithelium, and granular degeneration of the same.

"If this process advances to another stage, there occurs either a hyperplasia of the interstitial connective tissue, or, what is more frequent, a granular condition with atrophy. The cellular elements lying between the urinary tubules become converted into masses of connective tissue, which serve to obstruct the glomeruli and tubules. The increased blood pressure in the aorta induces hypertrophy of the left ventricle, and albumin appears in large quantity in the urine, which is increased in quantity, and of low specific gravity. According to all authorities, the abuse of alcohol is one of the most common causes of the granular kidney. According to Christison, three fourths or four fifths of all cases of granular atrophy are induced by it.

"The nervous system.—The affections of the nervous system in drunkards are both numerous and important. No organ, with the exception, perhaps, of the liver, suffers so constantly and from such a variety of lesions as the central nervous system. Many alterations in the functions are recognizable after death by a change in the tissues; but there are various affections, on the other hand, which point to a marked change in the cerebro-spinal system that cannot be detected. The very delicate and complicated structure of the nerves and ganglion cells requires not only that their anatomical, but also their chemical, relations shall be preserved for the performance of their functions. Ever so slight a deviation in the nutritive processes produces a disproportionate disturbance in their functions,—much greater than in any other tissues of the body.

"The brain.—The calvarium is altered. It is increased in weight by hyperostosis and sclerosis, both the outer and the inner table being

thickened. The cancellated structure is more dense, owing to a concentric formation of bone about the Haversian canals. Upon the inner surface, the channels of the vessels are deeper than normal, as well as the depressions for the Pacchionian bodies.

"There is an increase in the amount of blood in the brain, owing to the abnormal action of the heart and fatty or atheromatous degeneration of the walls of the small vessels, or diminished nutrition of the same, which paralyzes them so that their lumen becomes increased, and hyperemia results. In the earlier stages of alcoholism, where alcoholic excess is relatively frequent, this hyperemia is more of an active process, which, in the later stages, assumes a passive character, when obstruction to the circulation exists in other organs, as the liver, kidneys, lungs, etc.

"Cerebral apoplexy.—An effusion of blood into the brain substance frequently occurs in drunkards. All conditions brought about by the intemperate use of alcohol, which tend to produce cerebral hyperemia, favor, in a marked degree, the occurrence of either large or capillary effusions.

"Serous apoplexy.—An acute or chronic serous effusion into the cavity of the skull, into the brain substance, or into the membranes of the brain, and into the cavity of the arachnoid, may result from the abuse of alcohol. The transudation occurs as a complication in other cerebral diseases, and in those troubles which tend to produce hyperemia of this organ by mechanical stasis, as in diseases of the lungs and heart. It may also result from a very watery condition of the blood, such as occurs in Bright's disease. In alcoholism the blood is poor in plastic material, and as a consequence the transudation is favored. Either an acute or chronic collection of fluid in the ventricles of the brain is not an infrequent result of drunkenness.[76]

"Pachymeningitis interna chronica.—This inflammation of the inner surface of the dura mater consists, at first, of a very slight layer of fibrin on the surface of the dura, from which a thin layer of connective tissue is afterwards developed, which adheres to the surface of the membrane. A second and a third layer of inflammatory exudation is then formed, and so on until there are many layers. The dura mater thus becomes materially thickened. Each one of these layers is vascular and occasionally one of these vessels ruptures, resulting in a hemorrhage between two of the layers. That

[76] Hasse, Krankheiten der Nerven- und Therap. von R. Virchow, Bd. 4. s. systems. Handbuch der Spec. Pathol. 365.

this affection is more liable to occur in the intemperate, there can be but little doubt, although it has been but seldom produced in the lower animals, even after long and continued administration of alcohol."

473. **Action.**—As alcohol has the property of absorbing water and coagulating albumin, its local action upon the skin produces inflammation; the vapor of alcohol on the skin, protected from the air, will increase this inflammation; the local action on the mucous membrane is likewise irritating, causing nausea and vomiting. When absorbed into the general system, it still has the action of attracting water from the tissues, and precipitating proteids. The older theory of stimulation of the nervous centers has rather, of late years, been superseded by the theory that its action upon these nervous centers causes a diminution or inhibition of their functions. Its elimination is largely performed through the lungs, and a small portion through the kidneys, whose tissue it impairs; consequently, as it arrests metabolism of the tissues in small doses, not frequently repeated, it rather tends to retard tissue waste, and hence may be considered as a food substitute, but not a food. According to some theories of the modern chemists, alcohol is supposed to split up in the body into carbon dioxid and water; by others, to form a combination of true alcoholates of the chemical constituents of the brain, as in the formation of vegetable colloidal bodies which is seen in some fruits. Alcohol absorbed into the blood would rapidly pass into all of the tissues of the body, and even into the fetal circulation; in some instances it even passes into the milk of a nursing mother. Death usually occurs in acute poisoning by large doses of alcohol, from a paralysis of the nerve centers which incite respiration. Chronic poisoning from alcohol hardly has a place in a work on toxicology, but, rather, belongs to the realm of a treatise on the diseases.

474. **Lethal dose.**—On the authority of Kobert 2 to 6 ounces (60 to 180 gm.), but this varies according to the constitutional temperament of various persons, and age. We refer to cases of acute poisoning.

475. **Post-mortem appearances.**—In a case reported by Medical Examiner Draper (No. 135, in 1893), the following summary of an autopsy is returned by him: Heart filled with dark, fluid blood; mitral insufficiency; otherwise normal. Lungs congested and edematous. Tracheal mucous membrane red; no ecchymosis under skin of neck. Spleen, pancreas, and kidneys injected. Stomach and intestinal mucosa injected. Liver fatty, large, yellow, and dense. Brain wet; lateral ventricles filled with clear fluid. Slight odor of alcohol in organs.

Medical Examiner Draper, case 274, 1897. Ante-mortem symptoms of a child five and a half years old, who died from drinking 1 ounce (28.35 gm.) of raw whisky: Stupor, coma, convulsion, and death nine and a half hours later. Post-mortem appearances: Heart, numerous subpericardial ecchymoses. Blood dark and fluid. Old pleuritic adhesion. Lungs both congested; signs of bronchitis. Stomach empty, pale, normal. Spleen, pancreas, and intestines normal. Liver pale. Kidneys anemic. Mesenteric glands, large and hard. Bladder contained urine which gave alcoholic reaction. Brain injected.

476. Tests.—Alcohol warmed with sulphuric acid and a little potassium bichromate solution gives a green color and the peculiar fruity odor of ethyl aldehyd. Warmed with a little caustic potash and a bit of elementary iodin, alcohol forms iodoform, which can be recognized by its odor, and by the hexagonal, prismatic crystals seen under a microscope. A solution of molybdic acid in concentrated H_2SO_4 (1:10), when warmed with the suspected liquid, gives a blue color in the presence of alcohol, ether, or aldehyds. Shaken with benzoyl chlorid and soda solution until odor of benzoyl chlorid disappears, characteristic odor of ethyl benzoate develops. A blue color develops on adding guaiac, hydrocyanic acid, and copper sulphate. Upon heating ethyl alcohol with a solution mercuric nitrate, the mercuric salt is reduced, and a black precipitate results on adding ammonia; methyl alcohol does not give this reaction. Upon the addition of concentrated H_2SO_4 and a little sodium acetate, odor of acetic ether is observed.

477. Separation from organic mixtures.—All solid tissues should be finely divided, placed in a retort, and distilled over a water bath. Fluids, if at all acid, must be rendered neutral by adding a sufficient amount of sodium carbonate, then making slightly acid with tartaric acid, before being subjected to distillation. The fluid which distils over will contain the alcohol, more or less impure and dilute, so that it should be mixed with solid potassium carbonate and distilled again. This second distillate will usually have the odor of alcohol distinctly, and the amount of alcohol which it contains can be determined by placing it in a graduated vessel, and adding to it as much solid carbonate of potassium as it will take up; the alcohol will separate from the saturated carbonate of potassium solution, and form a separate layer at the top of the fluid. This upper layer of fluid consists of alcohol of 0.825 specific gravity, and contains 89 per cent of absolute alcohol. It can then be removed and subjected to the above-mentioned tests.

X. Amyl alcohol (fusel oil).

478. **In general.**—There are only a few cases of poisoning by fusel oil (impure amyl alcohol) after a single dose; but as this agent is found in many of the spirituous liquors, much of the chronic alcoholism has been imputed, whether justly or wrongly we do not pretend to discuss, to this form of alcoholism. Two cases of acute poisoning by fusel oil serve as the basis of an article reviewing the literature by Futcher.[77] The chief symptom described by him was unconsciousness, lasting several hours, and glycosuria which lasted two or three days. Glycuronic acid appeared in the urine, and in one case methemoglobin also. Experiments by Eulenberg[78] on rabbits, Cross[79] on pigeons, Rabuteau[80] on frogs, Furst on rabbits, and Sir B. W. Richardson[81] on various animals, would seem to show this a powerful poison,—especially when inhaled in its state of vapor; though Richardson expresses the opinion that fusel oil, when inhaled, is followed by peculiar symptoms lasting for several hours, and of such character that it might be impossible for the animal to recover; these symptoms are not shown to prove fatal. He observed muscular paralysis with paroxysms of tremulous convulsions. These spasms are the result of an excited reflex action by surface irritation, breathing on the animal, and in other ways subjecting it to trifling excitement.

479. **Post-mortem appearances.**—Its rapid inhalation causing death, the lungs and all the other organs are observed to be whitened and free from blood, the right side of the heart engorged with blood, the left empty, the brain being free from congestion. When inhaled slowly, the brain is found congested, and blood is found in both sides of the heart.

XI. Methyl alcohol (wood alcohol).

480. **Properties.**—Methyl alcohol, CH_3OH, wood alcohol, is formed in the destructive distillation of wood. It is a transparent, colorless, mobile, volatile liquid (sp. gr. 0.8142 at 0°; b. p. 66.5°), burns with a pale bluish flame, miscible with water, alcohol, and ether, in all proportions.

[77] Am. Med. Vol. II. p. 210.
[78] Gewerbe Hygiene, 1876, p. 440.
[79] De l'Alcohol Amlique et Methyl sur l'Organisme, (Thèse) Strasburg, 1863.
[80] Ueber die Wirkung des Athyl, Butyl, u. Amyl Alcohols, l'Union, 90 and 91, 1870, Schmidt's Jahrb. Bd. 149, p. 263.
[81] Trans. Brit. Association, 1864, 1865, and 1866. Also Brit. and For. Med.-Chir. Rev. Jan. 7, 1867, p. 247.

481. Occurrence.—Methyl alcohol, methylic alcohol, commonly known as wood alcohol, is in such common use that accidents and suicide have recently been quite common from taking this poison. Its use in paint factories, in washing windows, as a burning fluid, as a cheap form of alcohol for the solution of substances only readily soluble in alcohol, affords an easy means of obtaining this alcoholic poison. One case is offered as an example to show to what expedients men will resort when they desire the bracing effects of alcohol. Medical Examiner Bragg, of Massachusetts, reports (case 1, 1900) a fatal case of poisoning in a man who poured water into a solution of shellac made from wood alcohol, and drank the whole of the supernatant, clear liquid resulting from the precipitation of the resin by water.

By referring to the table of deaths, reported by the medical examiners of Massachusetts, in our first chapter, the reader will observe a constantly increasing number of cases of poisoning by wood alcohol in Massachusetts beginning with the year 1881, 38 fatal cases having occurred and been reported by the medical examiners during the last 22 years; of this number 1 is reported as a homicide, 27 as suicides, and 10 as occurring from accident or negligence. The earlier works on toxicology do not mention this as a poison. Peterson and Haines state that "poisoning with methyl alcohol is undoubtedly very common, but very few cases are reported in medical literature; in fact, almost no such cases were reported until recently, when the attention of the medical profession was directed to methyl alcohol as a cause of blindness. Apparently all these cases of wood alcohol poisoning, so far reported, have been due to accident; most of them have been caused by the drinking of wood spirit as a substitute for ethyl alcohol. In a number of cases, death or blindness has resulted from the use of extracts of flavoring agents that were made with methyl alcohol; such cases have been reported,—especially from certain 'prohibition' counties of Virginia and Maryland, and from the Indian territory[82] and Maine. Occasionally painters and varnishers are poisoned by the vapors of methyl alcohol." [83] Reference is made to the use of this alcohol as a solvent in the preparation of essences of Jamaica ginger, peppermint, cinnamon, lemon,[84] and of spirits of cologne and bay rum.

482. Symptoms of acute poisoning.—When death has followed the

[82] See Moulton, Jour. Am. Med. Asso. 1901, Vol. XXXVII. p. 1447.

[83] Patillo, Ophthalmic Record, 1899, Vol. VII. p. 599; Hale, Jour. Am. Med. Asso. 1901, Vol. XXXVII. p. 1450; De Schweinitz, Ophthalmic Record, 1901, Vol. X. p. 289.

[84] A number of such examinations

ingestion of a poisonous dose of wood alcohol, information gathered from the returns of the medical examiners of Massachusetts, and that given by other authorities, such as Moulton,[85] MacCoy and Michael,[86] describe the symptoms in a general way as follows: After the ingestion of poisonous doses of wood alcohol, the preliminary symptoms resemble those observed after an excessive dose of alcohol, such as exhilaration followed by hebetude and narcosis; but the severe headache, nausea, violent and persistent vomiting, are more marked. There are, also, abdominal spasmodic pains extending back over the region of the kidneys; there is also observed an ashy pallor of the skin accompanied with cold extremities, dilated pupils, great restlessness and exhaustion, weak action of the heart, syncope, and death within fourteen to twenty-four hours, when the amount of the poison is large. It would seem, however, that when the wood alcohol was taken by persons who were not habitual drunkards, the fatal action was prolonged for days, with continued coma.

Medical Examiner Draper, in his returns of case 301, 1893, to the secretary of state of Massachusetts, reports the symptoms of fatal poisoning by a man who drank by mistake for whisky, amount not stated, from a bottle labeled "Columbian spirits, not to be taken internally:" Abdominal spasmodic pains, also over renal region; ashy color of skin; cold extremities; dilated pupils; great restlessness and exhaustion; weak heart; syncope, and death fourteen hours after dose.

483. Symptoms of subacute poisoning.—The above authorities, as well as others,[87] describe the subacute symptoms which occur after twenty-four hours: In the meantime, the patient continues his work; the first symptoms to appear are chills and vertigo, to be followed by long-continued sleep, and in one case by death. Sometimes these symptoms are retarded for two days, and then there occurs a peculiar, disturbed condition of the vision, accompanied by dizziness, which gives indistinct outlines and confusion of colors. A single incident of this peculiar disturbance of the vision is offered by the experience of a patient who drank some wood alcohol, and afterwards, while unharnessing his horse, which he knew to be white, he noticed the

were made for methyl alcohol by Hyson and Dunning; see Ophthalmic Record, 1901, Vol. X. p. 83.

[85] See Moulton, Jour. Am. Med. Asso. 1901, Vol. XXXVII. p. 1447.

[86] MacCoy and Michael, Med. Record, 1898, Vol. LIII. p. 777.

[87] Kuhnt, Zeitschr. f. Augenheilk. 1899, Vol. I. p. 40; Thompson, Ophthalmic Record, 1897, Vol. 6, p. 579; Woods, *ibid.* 1899, Vol. VIII. p. 55; Harlan, *ibid.* 1901, Vol. X. p. 81; see also Wurdamann, Am. Med. Vol. II. p. 995, and Burnett, Therap. Gazette, 1901, p. 801.

animal to be red, but after a few days this distorted vision ceased. Gifford[88] reports the case of a man who lost his sight for three days, after drinking cologne spirits which contained methyl alcohol.

Individuals show a marked difference in susceptibility to methyl alcohol. Blindness and atrophy of the optic nerves have followed the taking of 2 to 5 drams (Raub); while recovery from a semicomatose condition, brought on by drinking ½ pint of methyl alcohol, without visual disturbance, has been recorded (Moulton). A few cases are reported where the inhalation of wood alcohol has caused blindness. In the subacute cases the symptoms observed are somewhat the same as in the acute poisoning, but less marked. The symptoms may be delayed for several hours. As a rule, those first noticed are nausea and vomiting, vertigo and headache. These may be followed by a period of coma or semicoma. Complete blindness, as a rule, soon develops. The pupils are dilated and irresponsive to light. This blindness may persist for several days, and then vision begin to return gradually. A few cases have been reported where vision has returned to practically normal.[88a] As a rule, the vision is permanently affected to a greater or less degree. According to De Schweinitz, blindness has been practically complete in more than 90 per cent of the cases recorded. Cases with impaired vision show contraction of the visual fields, with either an absolute central scotoma or a central scotoma for colors. Often a case will show great variations in visual acuity, before complete blindness sets in. The ophthalmoscope shows, in the early stages, as a rule, nothing, sometimes a congestion of the optic disc, or even a positive optic neuritis; in the late stages optic atrophy with diminished size of the blood vessels. The pathological condition underlying the visual defects is a matter of some dispute. Some observers claim that the primary seat of the trouble lies in the ganglion cells of the retina, and the lesions in the optic nerve are secondary. Others claim that the optic nerve is primarily affected. Very likely both structures are affected at the same time (Gifford).

484. Symptoms of chronic poisoning.—The blindness which may occur after poisoning by the use of methyl alcohol, whether the patient has drank the same or been exposed to its vapor, may clear up and return again in a few days, with the result of permanent blindness. The narcosis, which is preceded by long-continued sleep, may become more and more accentuated, passing into coma, and, finally, death within a week or ten days.

[88] Gifford, *ibid.* 1901, Vol. X. p. 342. 1902, p. 52; Gifford, Ophth. Rec. July,
[88a] Schrapinger, Arch. of Ophth. Jan. 1901, p. 342.

485. Treatment.—When the poison has been swallowed, the stomach should be washed out, and a stimulant medicine,—of course avoiding alcohol,—such as caffein and digitalis, should afterwards be administered; coma should be treated by warm bath immersions, quickly followed by cold affusions. It has been suggested that a diuretic should be administered to help in the elimination by the kidneys of the original poison, as well as a secondary product of the decomposition of the methyl alcohol,—formate of sodium,—of which we will speak later. For the blindness, ophthalmic surgeons have successfully used pilocarpin, potassium iodid, strychnin, and cathartics.[89]

486. Lethal dose.—This is somewhat larger than that of the ordinary ethyl alcohol. Five men each drank a tumblerful of wood alcohol, all of whom became ill; two of these recovered entirely; one lost the vision of one eye, and a partial injury to vision in the other eye occurred, the other two died within twenty-four hours. A smaller dose than the above has resulted fatally. Complete destruction of the functions of the optic nerves has occurred after swallowing two to five teaspoonfuls of methyl alcohol.[90]

487. Action.—The physiological action, according to Mayer,[91] and from experiments on animals by Pohl[92] and Birch-Hirschfeld, would seem to indicate that the ingestion of repeated doses of wood alcohol is followed by more serious and dangerous symptoms than is the case with ordinary or ethyl alcohol. Methyl alcohol is but partially oxidized in the body, while ethyl alcohol, being more readily oxidized, passes out more rapidly. Owing to the fact that the methylic remains longer in the tissues of the body and is not decomposed by oxidation, a secondary product of sodium formate is apparently formed, which is slowly passed out through the kidneys. The sodium formate is eight times more poisonous than methyl alcohol. This may account for the more serious symptoms occurring after the ingestion of wood alcohol than after ordinary alcohol,—especially as the same differential effects have been seen in cases of poisoning in man. This may also explain the delayed symptoms of poisoning by methyl alcohol, which have been observed in man after continued ingestion of repeated small doses.

[89] Kuhnt, Zeitschr. f. Augenheilk. 1899. Vol. I. p. 40; Thompson, Ophthalmic Record, 1897, Vol. 6, p. 579; Woods, *ibid.* 1899, Vol. VIII. p. 55; Harlan, *ibid.* 1901, Vol. X. p. 81; see also Wurdamann, Am. Med. Vol. II. p. 995, and Burnett, Therap. Gazette, 1901, p. 801.

[90] Raub, *ibid.* p. 619.

[91] See Mayer, Archiv. f. exper. Path. u. Pharm. Vol. XXI. p. 122.

[92] Pohl, Archiv. f. exper. Path. u. Pharm. Vol. XXXI. p. 281; Joffray and Serveaux, Arch. de Med. exp. 1896. Vol. VIII. p. 473; Hunt, Johns Hopkins Hospital Bulletin, Vol. XIII. p. 213.

488. Post-mortem appearances.—There are only a few cases of death from poisoning by wood alcohol in which the post-mortem appearances have been carefully reported in medical literature. Two of these are given in the medical examiners' reports to the Massachusetts secretary of state (Medical Examiner Titcomb, cases 1 and 2, 1892):—

Autopsy: Surface of body dark-colored. Heart, blood vessels on its surface engorged with dark blood; right ventricle full of liquid blood, dark-colored; left ventricle empty. Liver congested, and its vessels moderately filled with dark fluid blood. Lungs congested,—especially the lower lobes; pulmonic tissue firm to touch; inner walls of large bronchi covered with serosanguineous fluid, and their mucosa deeply injected. Kidneys congested. Intestines not remarkable. Stomach, internal surface deeply injected. Spleen dark in color. Brain, vessels of dura mater full of dark-colored blood; brain substance deeply congested with numerous extravasations. No other diseased appearance in any other organ.

489. Detection and tests.—Methyl alcohol can be isolated from the tissues as described under ethyl alcohol, and can often be recognized by its odor. Pure methyl alcohol does not give iodoform with caustic potash and iodin.

For special tests to differentiate it from ethyl alcohol, see tests under the latter.

XII. Benzol; benzene; naphtha; nitrobenzol; paraffin oil; and the benzol group.

490. In general.—Strictly speaking, the benzol group of poisons belongs to the class of anilin poisons, which we have previously considered (page 376), and the symptoms of which have been presented.

491. Properties of benzol.—Benzol is a colorless, mobile liquid, very sparingly soluble in water, soluble in alcohol, ether, and acetone, having at ordinary temperatures, when pure, a disagreeable odor (sp. gr. 0.86; m. p. 4.5°; b. p. 80.5°). It dissolves iodin, sulphur, phosphorus, rubber, resins, and almost all the alkaloids. It is highly inflammable, burning with a luminous, smoky flame. With bromin or chlorin it forms addition substitution products.

492. Nitrobenzol in general.—Artificial oil of bitter almonds. "Essence" or "oil of mirbane" is a yellow, sweet liquid with an odor of bitter almonds, almost insoluble in water, freely soluble in alcohol and ether. Boiling point 213°.

493. **Occurrence.**—Nitrobenzol (nitrobenzene, "essence" or "oil of mirbane," artificial oil of bitter almonds) is used extensively in the manufacture of anilin, of perfumes, of explosives, and as an artificial substitute for oil of bitter almonds in flavoring extracts. Accidents have occurred, and even fatal poisoning, from the inhalation of the vapor by spilling the nitrobenzene on the garments of the workmen. Almond glycerin soap is scented with nitrobenzene. It has also been used for committing suicide and to procure abortion. The symptoms of poisoning resemble so closely those of the anilin products that it is hardly worth while to repeat them. The resemblance of its odor to that of prussic acid has been previously mentioned. Small doses of this substance, when taken with alcohol, seem to exaggerate the symptoms of poisoning. Mortality in the cases of poisoning indicates that about 40 per cent are fatal. A part of the benzene appears in the urine as sulphuric-acid-phenol-ester; another part is exhaled.

494. **Symptoms.**—Small doses of nitrobenzol are poisonous when swallowed or inhaled. It was first mentioned by Casper, in 1856, as a poison.[93] The vapor is more poisonous by inhalation than is the liquid when swallowed.[94] Poisoning by this substance differs from that of prussic acid, in that the symptoms come on more slowly and are not so pronounced. They come on with a general feeling of lassitude, usually without convulsions. "The eyes are bright and glassy, the features pale and ghastly, the lips and nails purple, the skin clammy, and the pulse feeble." [95] After several hours the patient becomes unconscious, with stiffened muscles, followed by general convulsions. A fatal case[96] is reported by Mr. Nicholson. Taylor[97] reports a case of fatal poisoning which was the subject for an inquest in the Isle of Man, and another which was brought into the London Hospital. There were no immediate symptoms in the second, but the boy, aged seventeen, said that he felt drunk; and he became stupid and comatose, dying without the occurrence of vomiting or convulsions, twelve hours after he had swallowed the poison. Dr. Letheby reported another case, where death was caused by inhaling the vapor

[93] Vierteljahrsschrift, Bd. XVI. p. 1.
[94] Taylor, op. cit.
[95] *Ibid.*
[96] Lancet, 1862, I. p. 135.
[97] Op. cit. The reader may consult the following additional authorities: Christel. Zeitschr. f. Anal. Chem. Vol. XXIII. p. 94; Braun, *ibid.* Vol. IV. p. 185; Bondi, Prag. Med. Wochenschr. 1894, Vol. XIX. pp. 129, 143; Schild, Berlin, klin. Wochenschr. 1895, Vol. XXXII. p. 187; Ehlich und Lindenthal, Zeitschr. f. klin. Med. Vol. XXX. p. 427; Dodd, Brit. Med. Jour. 1891, Vol. I. p. 849; White, Prov. Med. Jour. 1892, Vol. XI. p. 462; Lancet, 1894, Vol. I. p. 1521; Marpurgo, Zeitschr. f. anal. Chem. Vol. XXXII. p. 235; Hoffmann, Liebig's Annalen, Vol. LV. p. 200.

of nitrobenzol, which was spilled over the wearing apparel of the victim. The symptoms were similar to those cases in which the poison was swallowed.

The delay in the appearance of the symptoms is not always of long duration. It depends upon the condition of the nitrobenzol. If the poison is taken in pure form, which is not soluble in water, and therefore does not mix with the fluids of the stomach, it is absorbed very slowly, and the symptoms may be delayed for many hours after taking the poison; but, on the contrary, if the poison be taken in spirit in which it is readily soluble, the symptoms may come on almost immediately after swallowing it. The blue, livid appearance of the skin and mucous membranes may appear some time before the patient feels very sick. Vomiting very frequently occurs, the vomitus having the peculiar odor of nitrobenzol, which resembles that of the bitter almond. The pupils are dilated and do not contract when exposed to a strong light. There is often pain in the stomach and abdomen, and incontinence of the urine and feces. In many cases, however, the blue color of the skin and the deep sleep are the only symptoms. Death may occur in four or five hours, but usually does not, in fatal cases, until after the lapse of from eight to twenty-four hours.

From Dr. Starkow's investigations,[98] it appears that the nitro-substitution products of these substances, if soluble, are more poisonous than the substances themselves, and produce a special action on the blood. The blood of an animal poisoned with dinitrobenzol, if subjected to examination by the spectroscope, shows an absorption band between the red and the orange in the spectrum, and corresponds to the Frauenhofer line *C*, in addition to the two normal oxyhemoglobin bands; reducing agents, such as ammonium sulphid, change the position of this band a little to the right, and ammonium hydrate causes it to entirely disappear. The blood of an animal, poisoned with (mono-) nitrobenzol, or nitro-naphthalene, shows the same band; but if these substances are mixed with blood outside of the body, the band does not immediately appear as in the case of dinitrobenzol, but only after the lapse of several hours. These mono-nitro-substitution products are less active poisons than the di-nitro-substitution products, but are much more active than the simple benzol, anilin, etc., or the chlorin substitution products, as chlor-benzol, none of which give rise to the above described absorption band; although

[98] Virchow's Archiv. III. No. 4; and 1873, abstract by the writer in Boston Jour. of Pharm. et de Chimie, April, Med. and Surg. Jour. July 10, 1873.

chlor-benzol, when mixed with blood, causes a disintegration of the blood globule, and the speedy appearance of hemoglobin crystals.

Letheby[99] records (as stated on a preceding page) a case of a man, forty-two years old, who spilled over his clothes some nitrobenzene, and, while engaged in his work for several hours, inhaled an atmosphere charged with the vapors of the poison. He became drowsy, staggered in his gait, and appeared intoxicated; he became sleepy and afterwards comatose, dying about nine hours after the first observance of the symptoms. Thompson[100] reports a similar case, where the nitrobenzene fumes were inhaled while transferring nitrobenzene into smaller vessels. The symptoms were dizziness, headache, vomiting, hurried respiration; he exhaled the vapor of nitrobenzene, the surface of the body had a dark purple color,—especially marked on the lips and under finger nails; heart action was hurried, and there was a tendency to sleep; urine dark in color, smelling strongly of nitrobenzene. Under treatment,—especially directed to assist the elimination of the poison by the lungs and kidneys,—the patient recovered in five days, but still retained his livid complexion.

495. **Lethal dose.**—Fifteen drops of nitrobenzene have proved fatal. Patients have recovered under treatment, after 24 drams (113 c. c.). Death has occurred within four hours after poison was taken, but usually the period is prolonged up to ten hours.

496. **Subacute poisoning.**—Many cases have been reported where exposure to the fumes of nitrobenzene in factories commonly causes symptoms of chronic poisoning such as languor, sleepiness, hurried respiration, cyanosis, and sometimes failure of the sight. The urine gives off the odor of nitrobenzene, somewhat similar to prussic acid, has a dark maroon color, and, on being shaken with chloroform, will extract and dissolve the nitrobenzene. The period of convalescence, under appropriate treatment in chronic poisoning, is about ten or twelve days.

497. **Treatment.**—When swallowed, the stomach should be washed out, and appropriate heart stimulants, avoiding alcohol, be given. The breathing of fresh air, artificial respiration, and oxygen should be employed after an acute attack of poisoning.

498. **Action.**—This appears to be similar to that of the anilin series; *viz.*, serious changes in the physical condition of the blood corpuscles, whereby metabolism of the tissues is obstructed, and the nerve centers which control the movements of the heart and respiration are paralyzed. By the spectroscope the peculiar absorption

[99] Op. cit. [100] Brit. Med. Jour. 1891.

bands of methemoglobin can be recognized, sometimes the appearance of another absorption band. The blood may contain only 1 per cent of oxygen, instead of 17 per cent as in health, and it appears incapable of taking up more oxygen when shaken with air. As a result of this blood poisoning, nephritis is usually found present after death from nitrobenzol. It would appear as if the nitrobenzol was changed into an anilin product, which can be recognized in the urine.

499. Diagnosis.—As in the case of poisoning by the anilins, by the peculiar absorption band of methemoglobin in the blood, and the production of the black anilin deposited in the tissues; also, by the smell of oil of bitter almonds, which it resembles.

500. Post-mortem appearances.—Its odor is very much more persistent post-mortem than is that of prussic acid. Drops of nitrobenzol may be found in the stomach and intestines several days after death. The œsophagus, stomach, and duodenum are spotted with ecchymoses. The blood is fluid and dark brown, the physical condition of the blood corpuscles is changed, and the blood is with difficulty coagulable. This is an important point in distinguishing nitrobenzol from prussic acid poisoning; in the latter, the blood is usually cherry-red in color. The blood also, when examined with the spectroscope, shows the absorption band of hematin, which is not present in normal blood.

501. Detection and tests.—The material under investigation is subjected to fractional distillation. The fraction boiling between 70° and 90° is again fractionated, and by successive distillations the benzol can be recovered almost pure. Various substitution products, and derivatives, such as anilin, can be made, which will serve to identify it.

Essential oil of almonds adulterated with nitrobenzol yields a blue color when distilled with lead acetate and chlorinated lime is added to distillate. A red color is produced on adding potassium hydrate, alcohol, and a little sulphur to nitrobenzol. On adding a solution of stannous chlorid in caustic soda, anilin is formed, and a blue color develops on adding carbolic acid to chlorinated soda solution. Carefully heat to boiling, in a porcelain dish, two drops liquefied phenol, three drops of water, and a small piece of KOH, then add liquid to be examined. On boiling, a crimson ring appears at the margin of the liquid. If, now, saturated solution of chlorinated lime be added, the ring becomes green.

The principal tests for nitrobenzol are: (1) Its powerful odor resembling that of bitter almonds; (2) its insolubility in water, so that

it floats upon water in the form of oily drops; it can easily be removed from such a mixture by shaking with naphtha, which readily dissolves it, and, upon allowing the naphtha to evaporate in a watch glass at the ordinary temperature, the nitrobenzol will be left as a residue; (3) it can be converted into anilin by being dissolved in alcohol, and this solution treated with a little metallic zinc and hydrochloric acid; the nascent hydrogen thus evolved converts the nitrobenzol into anilin, which can be separated by shaking the mixture with ether, removing the ether, and allowing it to evaporate, when the anilin will be left as a residue in the form of an oily fluid, which will react to the ordinary tests for anilin.

In legal cases the chemical examination enables us to readily distinguish between prussic acid and nitrobenzol. In the first place, in cases of poisoning by the latter substance the odor is perceptible for a much longer time than in prussic acid poisoning; and, in the second place, with this powerful odor we are unable to obtain the vapor tests for prussic acid, which would not be the case in prussic acid poisoning.

502. Naphthalene; properties and occurrence.—Napththalene is the simplest compound of those substances in which two or more benzol rings are united in such a manner that each two possess two atoms of carbon in common. It occurs in coal tar. It crystallizes in large shiny plates, fusing at 80° and boiling at 217°, but sublimes at lower temperatures. It burns with a smoky flame, is insoluble in water, soluble in alcohol and ether, has a burning taste and a faint aromatic odor. It forms substitution products with chlorin, bromin, iodin, and nitric acid.

503. Naphthalene; detection and tests.—The material under investigation is distilled with sodium, and naphthalene is found in the distillate, from which it can be extracted by shaking with ether. It can be differentiated from alpha- and beta-naphthols as follows:—

One part naphthalene dissolved in 25 parts of melted chloral hydrate remains colorless after ten minutes' warming on the water bath. Alpha-naphthol becomes red, beta-naphthol blue. If this mixture is then warmed with a little hydrochloric acid, a pale rose color may develop. The red of alpha-naphthol becomes greenish-blue, and the blue of beta-naphthol is changed to yellow. If zinc is now added, the naphthalene mixture becomes violet or brown, the alpha-naphthol blue-violet, and the beta-naphthol dark brown. Both the latter dissolve in alcohol with fluorescence.

A few drops of ammonia water or sodium hydrate solution causes fluorescence in a solution containing naphthalene.

If 3 or 4 drops of a solution of calcium chlorid, and a few drops of concentrated hydrochloric acid, are added to naphthalene, a lemon color develops. This is extracted with ether, and is overlaid on a 1 per cent aqueous solution of resorcin. A bluish-green color develops on addition of ammonia, which color is changed to cherry-red by nitric acid.

XIII. Disulphid of carbon.

504. Properties.—Bisulphid of carbon, carbon disulphid, carbon sulphid, is a colorless, mobile liquid with a peculiar odor; it boils at 47°, is very volatile, immiscible with water, but soluble in alcohol and ether. It is highly combustible, burning with a blue flame and yielding CO_2 and SO_2. It dissolves sulphur, phosphorus, rubber, fats, and oils readily. With iodin it forms a violet-red solution.

505. Statistics of poisoning by.—There are but few cases of fatal poisoning by this agent; one case, nonfatal, only reported by Mr. Blyth. This occurred as an attempt at suicide with 2 ounces (56.7 c. c.).

506. Symptoms of acute poisoning.—A fatal case of poisoning is recorded from a dose of 4 drams (15.55 c.c.). Foreman[1]:—Symptoms were pallor of the face, dilated pupils, frequent and weak pulse, diminution of bodily temperature, and spasmodic convulsions; death occurred in two hours and a quarter.

Experiments on animals have frequently been made, and carbon disulphid has been found to be fatal to all forms of animal life,—especially is this true as regards the destruction of various insects, such as moths, weevils in biscuits. It has also been used to exterminate mice and rats. Birds, rabbits, and guinea pigs are more susceptible to its poisonous action than either frogs or cats. Blood poisoning is shown to occur in animals by substitution of this agent for oxyhemoglobin by which the hemoglobin is reduced in the blood corpuscles. Kobert does not mention this substance as producing a combination with the hemoglobin of the blood. He does mention, however, that its inhalation causes headache, disturbance of vision, dizziness, vomiting, coma, and, with animals, convulsions. Its inhalation causes complete anesthesia of the whole body, and death in animals has been found to be caused by paralysis of the respiratory center, since artificial respiration fails to restore life.

[1] Foreman, London Lancet, 1886.

507. Chronic poisoning.—This occurs in factories where the carbon disulphid is used in large quantities for dissolving materials for making rubber clothing, for the solution of sulphur, phosphorus, oils, and resins. It is also used to disinfect apartments, by burning in a lamp. Thus there have occurred many cases of chronic poisoning from its inhalation. When persons are subjected to its inhalation for a long-continued time, a complicated series of symptoms have been noticed, the primary stage of which is excitement, with headache, disturbance of the digestion, its attendant loss of appetite, nausea, etc.; sensitiveness of the skin is also increased, with creeping sensations, numbness of the tongue, irritable disposition, ringing in the ears, complaint of noises in the head. In one instance, a workman in a factory suffered from a maniacal excitement, which subsided after a two days' removal from breathing the vapor. The sleep is disturbed by dreams, and pains in the limbs are a constant symptom, with spasmodic contractions of groups of muscles.

As a secondary set of symptoms, a stage of depression follows this pronounced anesthesia of skin. Patients complain also of a sensation as if the tongue was covered with a cloth. In this advanced stage, mental debility is very marked and the muscles are weak; sometimes paralysis of the legs, and, in one instance, paralysis of the right hand have been noted. Recovery after the first set of symptoms, above described, is generally quite rapid; but after the second set of symptoms a year has been known to elapse before any marked improvement occurred; a permanent injury to the health may be feared.

508. Lethal dose.—About ½ ounce (15 c.c.) has proved fatal, as in the case recorded by Foreman.

509. Treatment.—Stimulants and warmth to counteract collapse, artificial respiration and oxygen, of course removing the patient from all danger of inhalation of the gas.

510. Diagnosis.—By the unmistakable odor of exhalations from the lungs and from the body after death, on section.

511. Post-mortem appearances.—Except for the diminution of the hemoglobin in the blood corpuscles the lesions are not remarkable.

512. Detection and tests.—Extract the material with alcohol, filter, evaporate the filtrate, and apply the following tests: Its peculiar odor. A small bit of elementary iodin gives a violet-red color.

XIV. Camphor.

513. Properties.—The various camphors are white, odorous, vola-

tile, oxidation products, some of them aldehyds or ketones, and others alcohols. Ordinary camphor, Japan camphor, is a white, translucent, crystalline mass, with a strong, peculiar pungent odor and taste, slightly soluble in water, quite soluble in methyl and ethyl alcohols, ether, chloroform, acetic acid, and various oils.

514. Preparations.—(U. S. P.) Camphor water, containing about 1 part in 1,000; camphor liniment, containing about 200 parts in 1,000 parts of cotton seed oil; soap liniment, containing 45 parts in 1,000 parts of the solvent; spirit of camphor, containing 100 parts in 1,000 parts; camphorated tincture of opium (paregoric), containing 4 parts in 1,000 parts; monobromated camphor (monobromcamphor). There is also a strong solution (homeopathic) of camphor in spirits (Rubini's essence of camphor), equal parts of camphor and alcohol.

515. Symptoms.—Although camphor cannot be regarded as a very active poison, no well-authenticated case of death resulting directly from its use having, as far as we are aware, yet been reported, it is, nevertheless, capable of producing very dangerous symptoms. These, in the cases which are known, have varied somewhat, but in all there has been more or less evidence of its action upon the brain; vertigo, confusion of intellect, delirium, and somnolence being the most prominent effects. Indeed, the primary action of large doses of camphor is a powerful, but not a permanent, sedative of the nervous and vascular systems, followed by ataxic phenomena, and, remotely, by slight and very transient febrile excitement. Dr. Florain has reported the following curious case: A man, fifty-six years of age and of good constitution, took for the relief of priapism, and under a misapprehension of the directions of his physician, in enema 10 drams (39 gm.) of camphor. Immediately afterwards he had sensations of cold, alternating with heat in the lower bowels, and these sensations extended along the spine to the neck, and spread over the whole body. He was then seized with vertigo, had grotesque hallucinations, an excessive frequency of the pulse, embarrassed respiration, vomiting, and strangury, and was greatly prostrated within two minutes after taking the injection. The delirium increased, the features became pale and discomposed, the eyes fixed, and the pupils dilated. The skin became covered with clammy perspiration, and was ice-cold, the pulse frequent and thready, and the impulse of the heart very feeble. When violently aroused, the patient regained his consciousness for a moment, complained of distressing nausea, extreme chilliness, and great desire to sleep. Vomiting of a yellow, watery fluid,

smelling of camphor, followed, and was succeeded by great prostration. By the assiduous employment of stimulation, both externally and internally, as well as purgatives, the patient was rescued from this very precarious situation. He recovered entirely, and the only durable effect of the camphor was seen in the complete anaphrodisia which lasted for several weeks.[2] Two other cases are reported where the camphor was also given in injection of 1 dram (4 gm.), and which were followed by analogous symptoms. In one of these, the symptoms were very similar to those of an epileptic convulsion.[3] Dr. O. E. Brown, of Kentucky, mentions the case of a young man, who chewed and swallowed about 100 grains of camphor. No symptoms came on for a short time, but he was, perhaps an hour afterwards, suddenly seized with convulsions, and remained unconscious for several hours. He was relieved by bleeding and a warm bath. He gradually recovered his speech, but remained stupid, languid, and wandering all the next day.[4] A few cases are quoted by Drs. Taylor and Christison, in which camphor was taken by the mouth, but they do not differ essentially from the preceding.

516. Poisoning from use of.—Dr. Taylor[5] mentions three cases of fatal poisoning from camphor in infants under two years of age. In one of these camphorated oil was given by mistake; in another, half a teaspoonful of powdered camphor was followed by fatal results. Dr. Taylor also mentions several cases reported by Dr. G. Johnson, in which very dangerous, but not fatal, effects followed the use of a strong solution of camphor in alcohol, in small doses of 20 drops or more. This solution went under the name of "Rubini's homeopathic camphor," containing 50 per cent of camphor. It must, however, be admitted that very large doses of camphor may be followed by dangerous, but not fatal, symptoms.[6] Another case of poisoning by camphorated oil[7] is reported by Dr. Hewetson. Dr. Lamadrid[8] mentions a case in which an attack of acute gastritis followed an excessive dose of camphor.

The few cases of poisoning by camphor reported since the 4th edition of this work,[9] together with a review of the preceding cases, would seem to indicate, as in the case of the children, that the fatal effect was due to the alcohol rather than to the camphor; the youngest

[2] Gazette des Hôpitaux, No. 41, 1851.
[3] Canstatt's Jahresbericht für 1851, Bd. IV. p. 277.
[4] Boston. Med. and Surg. Jour. XXXVI. p. 868.
[5] Op. cit.
[6] *Vide* Trosseau et Pidoux, Traité de Thérapeutique, Paris, 1862, art. Camphre, action toxique, p. 267.
[7] Lancet. London, 1880–88.
[8] Phila. Med. Times, 1879, 325.
[9] Finley, Med. Rec. 1887, Vol. XXXI. p. 125; Davies, Brit. Med. Jour. 1887, Vol. I. p. 726.

child, of fifteen months, died in seven hours after taking a half teaspoonful of spirit of camphor. In another case, a woman took 184 grains (12 gm.) in a glass of brandy as an abortifacient resulting in death after abortion, within three days. In this case it is questionable whether the abortion or, not improbably, the large dose of brandy, would explain the fatal action following the camphor dissolved in the brandy.

There is not the slightest doubt of the fact that grave symptoms follow large doses of camphor, whether taken without solvent or in alcohol solution; but we fail to find a record of any death occurring to an adult except when the camphor was dissolved in alcohol.

517. Lethal dose.—The smallest dose which appears to have been attended with serious symptoms is 20 grains (1.3 gm.).[10] In a case related by Wibmer, as much as 8 scruples (10.37 gm.) of camphor dissolved in spirit were swallowed by a drunkard. This was followed by vertigo, dimness of sight, delirium, and burning pain in the stomach; there was vomiting, and yet the man recovered. The nature of the poisonous agent cannot fail, in cases where camphor has been taken, to be discovered, since the odor is so powerful and so well known as to betray itself at once.

518. Monobromated camphor.—Monobromated camphor, formed by the combination of bromin and camphor, is a violent poison in doses of 30 grains (2 gm.) or more.[11] The symptoms are muscular weakness, severe prostration, convulsions, great reduction of the bodily heat (sometimes to 30°C.), diminution in the number of respirations and of pulse, sopor, stupor, coma, and death. The symptoms have, at first, a rapid progress, but, when fatal, death takes place slowly. An insufficient number of cases of fatal results are reported to base any definite conclusions of pathological appearances. The drug has a very sedative influence upon the vasomotor nervous system, thus interfering with the functions of organic life,—especially the respiratory and circulatory systems.

519. Detection.—Camphor is readily detected by its powerful odor, and can be separated from organic mixtures by shaking the acidulated mixture with naphtha, which, upon being allowed to evaporate, will leave the camphor mixed with more or less fat.

This impure camphor can be purified by repeated recrystallizations from dilute alcohol (50 per cent), and then identified by its melting point, odor, taste, and spontaneous evaporation. If much camphor

[10] *Vide* Taylor on Poisons.

[11] Progrès Med. 1874; Practitioner, 1874–75; and a monograph by Pathault on Bromure de Camphre, Paris, 1875.

has been taken, the odor will be very perceptible in the contents of the stomach and intestines.

XV. Chloral hydrate.

520. Properties.—Chloral hydrate, wrongly called chloral in the U. S. P., is a white, crystalline compound formed when chloral, CCl_3COH, is mixed with water. It is slightly volatile at ordinary temperatures (m.p. 58°, b.p. 97.5°). It is easily soluble in water, alcohol, and ether, has an odor peculiar to itself, a disagreeable taste which may be disguised by dissolving it in an alcoholic drink. It is readily decomposed by alkalies into chloroform, water, and formates.

521. Statistics of poisoning from use of.—Mr. Blyth reports, in his table of ten years' mortality, 127 fatal poisonings from hydrate of chloral; 111 of these occurred from negligence or accident; 15 from suicide, and 1 from murder. In the returns of the medical examiners of Massachusetts for the twenty-six years, there are 35 cases of fatal poisoning reported, 1 of which was homicide, and 34 suicide; but there were none caused by accident or negligence. The collation in medical literature of cases of poisoning by Witthaus and Becker shows 70 accidental deaths from this poison, 22 suicidal, and one homicidal. During the earlier introduction (about 1869) of this poison as a medicinal agent, deaths from accidental overdoses were more common than during the later years. This is particularly noted in the returns of the medical examiners of Massachusetts, in which none during the last twenty-five years are reported as accidental. It should be remarked that, after its ingestion on an empty stomach, it is very much more rapidly absorbed than when the stomach has been subjected to continuous alcoholic excesses. In the latter condition, though earlier absorption may be delayed, yet, at a later period, the absorption may be relatively quicker; and this may account for the more frequent accidental deaths, where this drug was employed to relieve the restlessness attributable to chronic alcoholic excess, as larger and more frequent doses may be required to overcome the wakefulness of chronic alcoholism.

Probably there are almost as many cases of abuse of chloral drinking as there are of morphin eating, in which the long-continued use of chloral either ends in death, or the persons become fit subjects for the insane asylum: but this question is one which belongs to the alienist rather than the toxicologist.

522. Symptoms of acute poisoning.—The symptoms of chloral poi-

soning vary in different persons and under different circumstances. When administered to a patient in full health, who is not addicted to alcoholic excesses, there is, at first, a period of excitement of short duration, which should immediately be followed by a tendency to sleep. In moderate doses, below 30 grains (2 gm.), this tendency can be resisted, and the patient can ward off sleep; with the same dose repeated at too short an interval, or with larger single doses, the tendency to sleep is not controllable and has the character of narcosis. The bodily temperature falls below normal in a very few moments after taking the dose, ten to twenty minutes; occasionally, the preliminary excitement may be almost delirious before sleep is effected. During the deep slumber of narcosis, the face is flushed and the veins prominent; and in the case of an overdose, or several normal doses taken too near together, sleep may pass directly into death without any marked change. In some cases there may be a period of collapse, and the patient pass away in syncope. The time from the ingestion of the poison until the moment that death arrives is very variable, and is in part dependent upon the size of a single dose or a moderate dose repeated often—say once in an hour. The dangerous symptom that follows a single overdose of chloral is profound sleep, accompanied with stertorous breathing, not very unlike that produced by opium; but still the patient can be partially aroused, though unable to talk coherently; this profound sleep may pass into coma, muscular relaxation, and finally death.

523. Symptoms of chronic poisoning.—Both subacute and chronic poisoning present symptoms varying in character with individuals. After continuous use of chloral, the digestive system may suffer from disturbances complicated with diarrhea; usually these symptoms, if continued for a long period of time, result in emaciation. Dr. Smith calls especial attention to a rash and cutaneous desquamation which occurred in these cases.

The first symptom of danger, after the continued abuse of this drug, is a peculiar rash resembling in some particulars that of scarlatina or roseola. This may be followed by desquamation of the cuticle, and is generally, perhaps invariably, accompanied by albuminuria.

After subacute poisoning there may also be seen a purplish color at the roots of the nails, a still further proof of arrest of nutrition of the tissues. It is not uncommon to see these last-named symptoms of defect of nutrition follow as a sequela of a single large dose; but it is more usually observed after the continuance of several small doses. With the defective signs of nutrition, there may be associated

irritability of the nervous system, such as sleeplessness, general malaise, clouded intelligence; sometimes a depression of the spirits, almost melancholia, results. After the continued use of chloral, similar conditions may exist which simulate those noticed in mania a potu, when the use of the drug is suddenly suspended. Habitual users of chloral—especially when the drug is taken for a long period of time—may die suddenly; but this accident generally occurs from overstepping their usual medicinal dose.

524. **Lethal dose.**—In 7 cases of poisoning quoted by Mr. Blyth, three persons took a small dose, from 20 grains to 40 grains (1.30 to 2.60 gm.) respectively, and lived from eight to ten hours; two, taking 62 and 67 grains (4 to 4.3 gm.) respectively, died very shortly after the administration of the chloral; in a sixth case (related by Brown), 53 grains (3.5 gm.), the patient lived an hour; in another, after a dose of 78 grains (5 gm.), death took place in a quarter of an hour. The fatal doses recorded would seem to be, for an adult, between the two extremes, 30 grains to 1 ounce (2 gm. to 31 gm.). This latter would hardly be a safe dose, under our present knowledge, as derived from recorded cases. Any dose above 123.44 grains (8 gm.) would be dangerous, unless treatment should be promptly applied. Doses above 153.4 grains (10 gm.) may always be expected to be fatal. If, however, the 8 grams were administered in divided doses, four or five hours apart during the twenty-four hours, it would not be expected to be a dangerous dose. English physicians consider 20 grains (1.30 gm.) a very full dose for a child of four years, and 50 grains (3.24 gm.) for an adult. A young woman of twenty years was fatally poisoned by 30 grains (2 gm.), in 35 hours after taking the dose. While it may be said that children bear proportionately larger doses, according to their age, better than adults, old persons are more susceptible to the poisonous action of small doses. A child a year and a half old died in ten hours after taking a dose of 3 grains (0.195 gm.). When first used in France, early after its medicinal introduction, in order to produce anesthesia, a patient who received by intravenous injection a solution of 10 grams in 30 cubic centimeters of water became insensible before the whole quantity was injected. The patient was aroused immediately by the application of an electric current, one pole on one side of the neck, the other on the epigastrium. The injection of 6 grains (0.390 gm.) of chloral into the veins for the purpose of producing surgical anesthesia was followed by death in six minutes.[12] The introduction by rectal enema

[12] Chicago Med. Rev. 1882.

of 5.86 grams of chloral in a solution caused the death of a patient in three hours.

Another interesting case in Medical Jurisprudence is described by the patient himself:[13] He took in three doses, hourly, 185 grains (12 gm.) of chloral hydrate. The pain, which he, a physician, sought to relieve, vanished after the first dose; unwittingly, he took the other two doses, and sank into a sleep lasting twelve hours. When he awoke, he experienced difficulty in answering questions; he lay still, after sleeping for the next twelve hours, and subsequently slept all night, waking up recovered.

There have also been cases reported, where it was used with suicidal, but none with criminal, intent. Our notice will, therefore, be confined to a review of some of the cases which have been noticed in the various medical journals.

Dr. Smith, of Baltimore,[14] reports four cases which came under his personal observation; in some of these fatal cases of poisoning, chloral hydrate had been used in moderate continued doses, and in the others in an excessive single dose.

Dr. Richardson, of London,[15] considers 2 drams of chloral a dangerous, and 3 drams a fatal, dose. He thinks, also, that we cannot safely increase the daily dose (in fact it is in this way that many of the accidents have occurred) of chloral, as we can that of opium, and that the system does not become habituated by its continued use, nor able to eliminate larger doses than at first.[16]

A clergyman, in England,[17] had been in the habit of using chloral in small doses to induce sleep. Though the precise dose that preceded his death could not be ascertained, yet, during ten days, he had consumed from 14 to 15 drams (nearly 2 oz.).

In the first half of the volume for 1871 of the London Medical Times and Gazette are recorded three more cases of death occurring after the use of this drug.

Dr. H. W. Fuller,[18] reports a case in which 30 grains (2 gm.) of chloral produced alarming symptoms, and another in which the same dose produced death.

Dr. Needham[19] reports the case of a woman, aged fifty years, who took 30 grains of chloral to relieve a mental unquietness amounting

[13] Dr. Manjot, Gazette des Hôpitaux, 1875.

[14] Boston Med. and Surg. Jour. July, 1871, p. 33.

[15] Med. Press and Circular, Feb. 25, 1871.

[16] See Appendix.

[17] Brit. Med. Jour. Feb. 25, 1871.

[18] London Lancet, Mar. 27, 1871.

[19] The Med. Times, Feb. 15, 1871.

to insanity. The first day she took this dose at half past five in the evening and repeated it at eleven o'clock. The second day at ten in the morning she took 15 grains (1 gm.), and at three in the afternoon she repeated this dose. The third day she took 30 grains (2 gm.) at one o'clock in the morning, again a few moments after eight, and finally at half past one in the afternoon. On the evening of the second day she was out of bed and moving about. On the third day she was sleeping and had a somewhat rapid pulse, and at six in the evening she was still sleeping. The next morning her pulse was 108, and attempts to arouse her were of no avail. The pupils were a little contracted. After several ineffectual attempts to arouse her during the day, 1/10 of a grain (0.0065 gm.) of strychnin (an antidote recommended by Prof. Liebreich (was subcutaneously injected in three separate doses; but she passed from her lethargic sleep into a state of coma, and died the next day at four in the afternoon.

Dr. Dabb reports in minute detail[20] a case where serious apprehension was felt for the life of his patient.

Another case has been reported in the service of Dr. Ludlow[21] at the Philadelphia Hospital, where a nurse, supposed to have swallowed 460 grains (30 gm.), was found in an unconscious condition, from which she was with great difficulty rescued by vigorous flagellation of the surface of the body and by the application of electricity.

"The maximum quantity of chloral hydrate that can be borne, at one dose, bears some proportion to the weight of the animal subjected to its influence. The rule, however, does not extend equally to animals of any and every class. The proportion is practically the same in the same classes, but there is no actual universality of rule. . . . The human subject, weighing from 120 to 140 pounds, will be made, by 90 grains, to pass into a deep sleep, and by 140 grains, into fatal sleep. . . . Evidence has been brought before me which leads me to think that, although 80 grains would, in most instances, prove fatal, it could, under very favorable circumstances, be recovered from.

"Dr. Hills, of the Thorpe Asylum, Norwich, has, for example, favored me with the facts of an instance in which a suicidal woman took no less than 472 grains (31 gm.) of the hydrate of chloral dissolved in 16 ounces (480 c.c.) of water, and actually did not die for thirty-three hours. Such a fact, ably observed as it was, is startling; but it does not, I think, militate against the rule that 140 grains (9 gm.) is

[20] Med. Times and Gazette, Oct. 8, 1870.

[21] Med. Times, Oct. 15, 1870.

the maximum quantity that should, under any circumstances, be administered to the human subject. . . . As a near approximation to the truth, an adult person, who has taken chloral in sufficient quantity to be influenced by it, disposes of it at the rate of about 7 grains (0.5 gm.) per hour. In repeated doses the hydrate of chloral might be given at the rate of 12 grains (0.8 gm.) every two hours for twenty-four hours, with less danger than would occur from giving twelve times twelve (144) grains (9.2 gm.) at once; but I do not think that amount ought to be exceeded, except in the most extreme circumstances, even in divided doses."[22]

From other experiments and observations alluded to in the same paper, Dr. Richardson "has no doubt it will be found, as the chronicle of deaths from chloral hydrate increases, that the mortality of the agent will be greatest when the thermometrical readings are the lowest, and *vice versa.*"

As stated above, in acute alcoholism, or mania a potu, the absorption of chloral hydrate is slow or is retarded; this sometimes leads to fatal accidents from the incautious use of large doses. As the elimination of alcohol is accomplished, the accumulated large doses of chloral in the stomach may be all at once absorbed, and bring about a fatal poisoning.

From what is known with regard to the use of this drug, it is probable that not more than 20 to 30 grains of chloral should be administered in a single dose, and that it is somewhat dangerous to repeat this at shorter intervals than six to eight hours. (The absorption of chloral by a fasting stomach is quite rapid.)

525. Treatment.—Liebreich, who introduced chloral hydrate as a medicine, relied upon subcutaneous injections of strychnin as a special antidote to chloral poisoning.

Strychnin is not to be relied upon[23] as an antidote to chloral poisoning. The most theoretical and practical method of relief in cases of poisoning would be external stimulation, rectal injections of some stimulating liquid, as a mixture of castor oil or broth with turpentine; and the use of electricity, either in the form of galvanization or faradization; and also the placing of the patient in a warm atmosphere, and causing him to inhale warm air.

The absorption of chloral is so rapid that it seems almost impossi-

[22] The above is extracted from an exceedingly interesting report on the physiological action of the organic chemical compounds, by B. W. Richardson, M. D., F. R. S.

[23] *Vide* Gaz. Hebd. de Médecine et de Chirurgie, June 21, 1872, p. 409, and July 1872, p. 457.

ble to get rid of the poison by washing out the stomach, as in cases where other poisons have been swallowed; time should not be wasted in useless endeavors for this treatment. Artificial respiration should be tried immediately, also the inhalation of oxygen, and the application of hot woolen covering to increase the surface temperature of the body; apomorphin, by subcutaneous injection, often fails to cause vomiting, and will only add to the great depression already existing in the nervous system. A weak alkali given to the stomach may cause a decomposition of chloral by the production of chloroform, though we have not much confidence in this chemical decomposition inside the body. Hot coffee is a good stimulant for the heart, if the patient can be made to swallow it. We must confess that the methods of treatment should be practised under the direction of a skilful physician, except for the application of heat to the surface of the body to promote circulation in the capillary blood vessels. An unprofessional person, in trying to relieve a person poisoned by chloral, may be apt to attempt a treatment which would do more harm than good.

526. Action.—When first introduced as a medicinal agent, the theory of its action, as advanced by Liebreich, was to demonstrate in the body what can be accomplished in the chemical laboratory. Chloral in the presence of a weak alkaline solution is there decomposed into chloroform and formic acid. With the expectation of the absorption of chloral hydrate into the alkaline blood circulating in the body, Liebreich assumed the chloroform to pass into the tissues and produce anesthesia. At the time of the advancement of this theory, or shortly after, the present writer and many others disproved this theory of the decomposition of hydrate of chloral into chloroform within the living body, by experiments on animals.[24] Since then, writers on toxicology have abandoned this theory of Liebreich. While acknowledging that chloral hydrate can be decomposed in some degree by the blood outside the body at normal temperature, this action must be prolonged for several hours in order to furnish sufficient amount of chloroform to produce anesthesia. It has been found that a 1 per cent solution of alkali does not decompose chloral in sufficient amount at a blood heat, within the time in which the effects of chloral are shown in the body. It is more probable, as now admitted, that chloral hydrate is absorbed, and circulates as such in the nerve center which controls the heart's action; also, through the same inhibition of this nerve center, the respiration is slowed and finally

[24] N. Y. Med. Jour. 1872, Vol. XV. pp. 606–623.

ceases. If the theory of Liebreich were true, *viz.*, that chloral hydrate is decomposed by the alkaline condition of the blood, in circulation of a living animal, into formic acid and chloroform, we should expect the lethal action of formic acid to be displayed in the animal economy, which has been shown in discussing formic acid (p. 403) as a poison, in doses swallowed of less than 20 to 30 grams.

Chloral hydrate, whose action we have above indicated as rather belonging to itself than to any decomposition, is found in the urine in the form of urochloral acid; but the amount obtained of this acid does not account for all the chloral hydrate which has been ingested.

527. Diagnosis.—From the symptoms above named, and from the smell of chloral.

528. Post-mortem appearances.—These are stated by Dr. Francis Ogston[25] to be as follows: The blood vessels of the membranes of the brain are full of blood; the sinuses contain clotted blood; the arachnoid (serous) membrane is edematous; under this membrane, and in the ventricles of the brain, clear serum in considerable quantity is found; the brain substance appears somewhat shrunken, but no unusual number of hemorrhagic spots may be noted. The œsophagus is usually slightly reddened and thickened in folds, and its mucous membrane is softened. The mucous membrane about the larnyx is edematous, with fine injection of its blood vessels; the lungs are edematous and deeply congested. The right side of the heart is engorged and the left comparatively empty; the blood in the heart may be partly clotted and partly fluid; the rest of the blood-making organs, such as the spleen, liver, etc., will be filled with dark blood. In other words, the post-mortem appearances are those usually found after death from narcotico-acrid poisons, and when death may be assumed to have been caused by asphyxia.

The fatty degeneration and the infiltration of the kidneys, discovered in animals that have been killed by the administration of chloral, are probably due to the urochloral acid whose passage through the kidneys may induce this condition on account of the local action of this substance.

529. Detection.—By far the best method for its detection is that described under chloroform.

XVI. Chloroform.

530. Properties.—It is a heavy, colorless, inflammable liquid, vola-

[25] Edin. Med. Jour. Oct. 1878.

tile at ordinary temperatures, boiling at 61°. It has a burning, sweetish taste, neutral reaction, and a characteristic, sweetish, ethereal odor, sparingly soluble in water, but miscible with alcohol and ether in all proportions. It is frequently contaminated with alcohol, ethylchlorid, ethylaldehyd, chlorin, hydrochloric acid, and other impurities; and when these are present, it is generally more poisonous.

531. Poisoning from use of in general.—Cases of chloroform poisoning are, for the most part, due to an over inhalation of its vapor in anesthesia, and so can be considered accidental. It has occasionally been used for suicidal purposes, but we find but one case of homicide.

532. Statistics of poisoning from swallowing chloroform.—Falk records, as his examination of the records in medical literature, 27 cases of poisoning from swallowing chloroform, of which 15 were men, 9 were women, and 3 were children; 18 of the cases were suicidal, of which 10 died; the other 9 cases were accidental. Mr. Blyth gives in his table 103 deaths during the ten years, 88 of which were accidental, 14 from suicide, and 1 case of murder. (See also p. 20.)

533. Acute symptoms after swallowing chloroform.—The effects of chloroform taken internally appear in from ten to twenty minutes, and are very similar to alcoholic intoxication; but these only precede the more serious symptoms of coma, complete insensibility, stertorous respiration, convulsions, and finally death in a few hours. Most generally, however, the patient slowly recovers his consciousness, but is left with a pain in the throat and stomach, and often has jaundice, cough, and a slight catarrhal bronchitis. These symptoms are those related by Tardieu and Taylor, each of whom gives illustrative cases.

Poisoning by the internal administration of chloroform is totally distinct from the effects produced by its inhalation.[26] It is not impossible that a portion of the chloroform taken into the mouth may be inhaled before it is swallowed, and we may then have the symptoms of inhalation, as well as those due to the ingestion. The quantity of chloroform necessary to occasion serious symptoms is variable. One dram of the liquid chloroform has brought on narcosis followed by death, in the case of a boy four years old.[27] In another case of a private[28] in a cavalry regiment, 2 ounces were swallowed, causing serious narcotic symptoms. The stomach was washed out by means of a stomach pump, twenty minutes after taking the drug, and then ammonia was injected. This man recovered.

A lady, weakened by miscarriage, was affected, within five minutes

[26] Tourdes, Gaz. Hebd. de Médecine et Chirurgie, Paris, 1866.

[27] Taylor on Poisons, p. 741.

[28] Amer. Jour. of Med. Sciences.

after taking ½ ounce of chloroform, with convulsions, insensibility, dilated pupils, trismus, flushed face, a full and oppressed pulse, and foaming at the mouth.[29] Another person, also a female, swallowed 2 ounces of chloroform; there was a deep stupor, without congestion of the face, and the pupils were contracted.[30] In neither case was the pulse reduced. In a third case the symptoms were the same as in the second, and were also produced by 2 ounces of the liquid.[31] When death has been produced by the internal use of chloroform, its local irritant action has evidently been the chief cause of the fatal result. Three cases, at least, of this description are recorded.[32] In all of them the quantity taken was from 1 to 2 ounces, and the local symptoms were those of an active irritant of the stomach. In one case the air passages shared in the irritation, and their congestion was the immediate cause of death. In all, examination of the bodies showed softening of the mucous membrane of the stomach, and in one case ulceration also.

The details of these symptoms may be described in two classes: First, the local irritation upon the stomach and the air passages, as well as in the throat and abdomen, and vomiting with involuntary emptying of the bowels; and second, the absorption by inhalation in the lungs of the chloroform in the general circulation, which are not unlike those symptoms attributable to the usual inhalation of chloroform. After small doses there may be a period of excitement; after large doses a mental depression begins, immediately followed by unconsciousness, generally within ten minutes; the skin assumes a muddy color, is cool, and the respiration becomes irregular and stertorous; the heart's action is weak. When most of the chloroform is passed rapidly into the stomach, and not inhaled, death seems to be caused by the shock due to the gastrointestinal irritation; while, if the larger portion has been involuntarily inhaled, the pulmonary symptoms prevail. The mortality appears, from cases recorded by Elliott and Hirsch,[33] to be somewhat between the limits of 26 to 41 per cent in a total of 74 cases.

534. Subacute symptoms after swallowing chloroform.—After swallowing, if death has not occurred during acute poisoning, and the patient continues to live, he will suffer from coma for many hours, and, on coming out of the unconsciousness, from abdominal pain, bloody

[29] Med. Times and Gazette, Dec. 1857, p. 615.

[30] Annuaire de Thérap. XVIII. 55.

[31] Am. Jour. of Med. Sci. Oct. 1857, p. 367.

[32] Month. Jour. of Med. Sci. Vol. 77; Phila. Med. Exam. Nov. 1856, p. 659; London Lancet, April, 1859, p. 400.

[33] Med. Rec. 1885, Vol. XXVII. p. 29; Zeitschr. f. Klin. Med. 1894, Vol. XXIV. p. 190.

diarrhea, swelling of the liver, jaundice (icterus), and painful urination. If the primary irritation is due to most of the chloroform passing into the stomach, he may die from conditions of gastritis, and inflammation of the small intestine, or from failure of the heart's action; if a larger portion has been inhaled, he will die from predominance of the pulmonary symptoms, or from the failure of the heart. Either of these two conditions may persist for several days, after which the patient will slowly recover, the symptoms gradually disappearing.

535. Chronic poisoning from swallowing chloroform.—This may also occur from drinking small doses of the drug, the habit being instituted first for the relief of pain, sometimes as a substitute for some other soothing or narcotic remedy. In these cases, the chief symptoms are disturbances of the digestion, and also of the sympathetic nervous system, as well as from the other effects of narcotic poisoning, such as hallucination, or delirium tremens in alcoholism.

536. Lethal dose.—This is difficult to state because part of the drug may be swallowed and the other part inhaled; 1 dram (3.8 gm.) caused the death of a child of four years, and another of the same age recovered from twice this amount. An adult died after 4 drams (15.5 gm.). In one case a man, forty-three years of age, swallowed a dose of 1 ounce (29 gm.) and died within thirteen hours (Bridgman).[34] On the other hand, a man, forty-six years of age, drank 5 ounces (145 gm.) of chloroform, and vomited freely; on being taken to the hospital, the stomach was washed out, but the contents contained no odor of chloroform. The exhalations of the lungs contained chloroform. Efforts to breathe having ceased, artificial respiration was resorted to, which relieved the cyanosis, and pallor of the skin, and dilatation of the pupils; it also restored the reflex action of the conjunctiva, and the patient began to breathe naturally in a few moments. In four hours consciousness returned, but he complained of pain in the throat and belly. He left the hospital two days later apparently well.[35]

537. Poisoning from inhalation of chloroform.—Up to the beginning of the year 1867, the total number of recorded deaths by chloroform inhalation was 133, in 33 (25 per cent) of which, no possible explanation of death, in the state of our present knowledge, could be given, other than that of poison.[36] "The rate of mortality

[34] Bridgman, London Lancet, 1897, Vol. II. p. 384.

[35] Glasgow Med. Jour. 1898, Vol. XLIX. p. 347.

[36] Am. Jour. of Med. Sci. Oct. 1867, p. 324.

is as 1 in 3,500 administrations of chloroform (I really think it is as 1 in 2,000 to 3,500); we have no other remedial agent which approaches chloroform in point of danger."[37] Since that time the number recorded in the various medical journals of death which could be imputed only to the use of chloroform inhalation is very considerable.[38] Up to the time of the fourth edition of this work (1883) the number of unavoidable deaths during or immediately after the chloroform administration had been largely increased. Unlike ether and certain other anesthetic agents, death by chloroform may occur without asphyxia, and without any apparent warning, by syncope either from arrest of the heart's action or from interference with the movement of the blood through capillaries in the nerve centers, chiefly in the brain. This explanation is shown to be correct in a large number of cases, from the fact that recovery from syncope often occurs by starting up this arrested capillary circulation by electrical stimulation, or by lifting the patient up by the heels and allowing the head to hang down, thus by gravitation inviting a flow of blood in the depending portions of the body, and thereby restoring its suspended functions. A careful examination of such cases as these can leave no doubt upon the mind that death was attributable solely to the inhalation of chloroform, and that it may occur with a celerity unparalleled by any other poisonous agent whatever.

From January, 1881, to February, 1882, there occurred in the British Isles 9 deaths from chloroform, 5 of which occurred in five weeks, 4 from ether, 1 from a mixture of ether and chloroform, and 1 from ethylidene. In 2 of the deaths from ether the kidneys were diseased. Of the chloroform deaths, only 2 occurred after severe operations.

A case of death from chloroform occurred at the Royal Infirmary, Edinburgh, under Geo. B. Silke, House Surgeon, January 3, 1880, during an operation for removing a portion of bone from a finger which had been chopped off. Before the operation the patient had eaten a hearty meal, as was shown by the autopsy, which also revealed extensive pleuritic adhesions of left lung, liver much enlarged and

[37] B. W. Richardson, M. D., F. R. S., London Med. Times and Gazette, 1870.

[38] *Vide*, also, Medical News and Library, Phila. Vol. XXVII. pp. 89, 103, 118. Medical Times and Gazette, London, June, 1869, p. 695; Nov. 1869, p. 579. N. Y. Med. Jour. X. p. 194. Brit. Med. Jour. 1878, II. 699; 1879, I. 307; II. 94; 1880, I. 613. Australian Med. Jour. 1878, 170. Med. Times and Gazette, London, 1878, II. 465; 1880, I. 613. Lancet, 1878, II. 571, 862; 1880, I. 268, 346, 825, 973; 1880, II. 376, and 1882, I. 378. Edin. Med. Jour. 1879–80, 1007, 1014. Canada Lancet, Toronto, 1879-80, 349. Buffalo Med. and Surg. Jour. 1879–80, 531. Phila. Med. News, 1882, 295. Medical Press and Circular, 1882, 124.

fatty, though the heart was not excessively distended because the external jugular vein had been opened in hopes of relieving the patient; the blood was fluid, having an odor of chloroform. Out of 121 under chloroform, the period at which death occurred was as follows:—[39]

For intended operations, or immediately before	54
During operations	42
After, immediately, or soon	25
	121

Sansom[40] presents some facts in 100 cases of deaths caused by inhalation of chloroform, as follows: 44.6 per cent occurred before the full narcosis had been reached; 34.7 per cent during the second stage of anesthesia; 20.6 per cent shortly after this stage.

Kappeler mentions that, out of 101 cases of deaths from chloroform inhalation, 47.7 per cent occurred before the full anesthesia; 52.2 per cent during complete anesthesia.

It is generally admitted that chloroform inhalation should be caused by chloroform arranged so that the patient shall breathe 96 per cent of air.

It is fair to assume[41] that the extensive use of chloroform inhalation in surgery varies in mortality from 1 per 10,000 to 6.3 per 10,000. though it is quite probable that, in many of the cases where death has been reported as due to a shock during an operation, the true cause may have been overlooked, and really should have been attributed to the chloroform inhalation. In the Massachusetts medical examiners' returns, the deaths from chloroform inhalation were 39, 6 of which occurred from accident or negligence, and 33 were suicidal. One of the cases by accident is specified in the returns as being due to self-administered inhalation by patient in bed, the chloroform having been inhaled for neuralgia. Another, holding some chloroform in her mouth to relieve neuralgia, suddenly laughed hysterically, inadvertently swallowed it, and immediately died. Of the cases of suicide, two were cases where chloroform was swallowed by the mouth; in one instance, 1 ounce (31 c.c.), drank at one swallow. The third case (Medical Examiner Paddock, case No. 12), an old man of seventy years swallowed the contents of two bottles, one of which contained laudnanum, chloroform, and ammonia; and the other ½ ounce tincture of aconite. The contents of the first bottle were taken shortly

[39] Brit. Med. Jour. Feb. 18, 1882, from an article on Chloroform by E. H. Jacobs, M. D.

[40] Chloroform, Its Action. London Lancet, 1865.

[41] Blyth, op. cit. p. 149.

before breakfast time on an empty stomach, and the ½ ounce tincture of aconite immediately afterwards. He vomited some matter having the smell of chloroform; after violent retching he became unconscious, and died two hours after. Before death, the pupils were contracted and the breathing stertorous. These symptoms would indicate that this death should be excluded from that of suspected chloroform poisoning, on account of the predominance of the opium narcosis. The autopsy, four hours after death, showed that the chloroform had produced local irritation of the stomach and small intestine, while the other post-mortem appearances, as, for instance, the engorgement of the pulmonary vessels, serous effusion under the arachnoid membrane of the brain, cerebral ventricles empty, and the surface of the brain substance injected, would indicate poisoning by opium.

When inhalation may be injurious.—Anesthetic agents, now so much used in surgical, dental, and obstetrical practice, in the form of vapor, are, as is well known, capable of producing fatal effects. Occasionally death has been due to their maladministration, the patient being either unfitted to respire them, or having been required to inhale them unmixed with atmospheric air, or for too long a time. Such accidents have been extremely few in the case of ether, and, since the proper mode of its administration has been understood, that is, since care has been taken to admit a sufficient proportion of atmospheric air along with the vapor into the patient's lungs, there is not a single authenticated example of its having destroyed life. Chloroform, being more energetic and rapid in its action, has so frequently been the evident cause of death, that the operator cannot be too careful to ascertain its purity, and the probability of his patient being able to bear it, and to see that he does not inhale it to the exclusion of the atmospheric air, or for too long a time, or to a person not lying flat.

Notwithstanding every precaution, however, occasionally death will suddenly occur in the most unexpected manner from its inhalation. This has occurred in the practice and under the supervision of the most eminent surgeons. Many cases have been published, which it would, however, be tedious to enumerate.[42] We append, however, a few by way of illustration. Patrick Coyle, chloroformed for fistula; he inhaled for about a minute and almost instantly expired.[43] Abbey Pennock inhaled about 3 drams in two applications,

[42] Dr. Crisp laid before the Medical Society of London a table which he had made of the recorded deaths from chloroform up to June, 1853. They amounted to 42. At the end of 1859, they had already exceeded 60 in number.

[43] Dr. Warren, Effects of Chloroform, etc., Boston, 1849.

to relieve the pain of toothache, and died almost immediately after the second application.[44] John Griffiths had chancres and hemorrhoids; inhaled about 3 drams (12 gm.), and died in about ten minutes, during the incision of the hemorrhoids.[45] In the case of Madame Labrunne, related by M. de Confevron, the fatal effects were manifested in eight seconds, and the operator remarked constant winking of the eyelids. The patient repulsed the dentist's hand, making signs that the effect was not complete. She then made four or five fuller inspirations. At that instant, M. de Confevron removed the handkerchief, and took his eyes off her only for the instant occupied by placing it on the table; but in this brief instant he found the patient's face turned pale, the lips discolored, the features altered, the eyes turned upwards, the pupils horribly dilated, the jaw closed, the head drawn backwards; the pulse could not be felt; the limbs were all relaxed, and a few inspirations at long intervals were the only indications of life.[46]

538. Symptoms of anesthesia or etherization.—The external phenomena of etherization, whether produced by chloroform or ether, are very nearly alike. There is usually, at first, a little cough, with expectoration of mucus and a flow of saliva, and some laboring of the respiration; then the inspirations become strong and deep, and take place without difficulty; the pulse becomes quickened, and the eyes injected. With these early symptoms there are often irregular movements of the limbs, and expressions of various kinds are uttered; sometimes a patient will try to put away the sponge or instrument used, but more generally he is anxious to retain it. If the inhalation goes on, the face generally becomes flushed, the eyes are brilliant, and turn in different directions, often upwards; soon the eyelids droop; very often laughter and incoherent expressions are heard; the pulse begins to be slower, and a general insensibility with muscular relaxation follows. This is the true surgical period of anesthesia. If this period be passed and etherization be pushed to its utmost limits, the respiration becomes stertorous, the face livid, the pulse slow and weak, and death may take place with symptoms of asphyxia. To sum up, temporary excitement, then stupefaction or disorder of the intellectual powers, insensibility, complete muscular relaxation, and

[44] *Ibid.*

[45] *Ibid.*

[46] Med. Gazette, Vol. IX. 1849, p. 295. *Vide*, also, Med. Times and Gazette, Oct. 1852, p. 361; April, 1853, p. 369; Aug. 1853, p. 173; Oct. 1853, pp. 407, 422, 461; Nov. 1853, p. 562; Dec. 24, 1853. p. 665. Lancet, June and Oct. pp. 523, 410. Edin. Monthly Jour. April, 1850, p. 377; 1855–56. I. p. 524. Med. Gazette, Vol. IX. 1849, p. 295.

death, are the great and observable stages of etherization pushed to its utmost limits.[47-8]

Sometimes death may take place, as before remarked, without any warning symptom, the pulse and respiration suddenly ceasing, while the complexion and expression of the patient's face is perfectly natural; these cases are more apt to occur when the chloroform is administered to a person erect or in a sitting posture.

539. Action.—In discussing the action of chloroform, it must be expressly understood that we are speaking of pure chloroform having no impurities, which are sometimes present, such as alcohol, ethyl chlorid, aldehyd, ethylidene, chlorin, hydrochloric acid, and sometimes others. The poisonous action of chloroform inhalation would be increased by the presence of these impurities. Gerlinger[49] calls attention to the decomposition of chloroform in the presence of a flame near by; if this condition should exist for quite a while, the products of decomposition are very irritant, and when inhaled, produce fatal poisoning.

The local action upon the tissues of the body, whether without or within, is that of irritation. This local action also arrests ciliary motion in the mucous membrane brought in contact with the vapor; it also arrests the reflex response in the nervous system, whereby there may be a rise of arterial blood pressure and the retardation of the heart; this latter action may be present only for a short time. When chloroform is absorbed into the blood circulation, either by inhibition of the nerve centers from which reflex action responds, or from stimulation of the sympathetic nerve system, there is a period of excitement, followed immediately by a stage of depression. These symptoms, following the absorption of chloroform in the blood, affect first the intelligence, then the center of motion in the spinal cord, and lastly, the center of reflex action in the medulla oblongata, which controls the central organ of pulsation, *viz.*, the heart, and of the respiration by the lungs. In consequence of this action at the nerve centers, respiratory movements become less frequent and the heart beats become weaker and slower.

Kobert calls chloroform a protoplasmic poison whose specific action disturbs the protoplasm of the ganglionic cells, those of the brain, at first; afterwards, it dissolves the blood corpuscles and produces degeneration of all tissues. In consequence of this arrest of action of the protoplasmic cells, the results of the suspension of chem-

[47-8] Brit. and For. Med.-Chir. Rev. Jan. 1852.

[49] Archiv. f. exper. Path. u. Pharm. 1902, Vol. XXXXVII., p. 438.

ical action in the tissues will reduce the body temperature; also, from the same reason, fatty degeneration will occur in the cells of the liver, in the tissues of the heart and kidneys, from repeated or long-continued presence of chloroform in the blood, and even, sometimes, from a single prolonged administration.[50] According to Jumelle, plants placed in the vapor of chloroform appear to be insensitive to the stimulus of light. Isolated animal cells (leucocytes), exposed to the vapor of chloroform, lose spontaneous movement; many micro-organisms (bacteria) cease to multiply in contact with water charged with chloroform vapor (Blyth).[51] Volatile anesthetics suspend the excitability of the gray and white nerve substance of the brain, and this action is also shared by morphin. After the suspension of the excitation of the brain, when chloroform passes into its tissues, blood pressure in the arterial system falls, and the heart beats are slower and weaker (Kobert). Unlike ether, the inhalations of small quantities of chloroform, absorbed in a deep inhalation, may be rapidly poisonous. Where there is a fatty heart, atheromatous arteries, in diabetes, in chronic nephritis, and with chronic alcohol drinkers, the danger from administration of chloroform by inhalation is rendered more serious, and in these diseases the effects of chloroformization may be postponed until several days after recovery from the first inhalation. It should be inferred from this last condition, of the health of the person inhaling the chloroform, that frequently deaths are attributable to inhalation which are more reasonably attributable to other conditions of health prior to the inhalation of the chloroform.

540. Medico-legal bearings.—Dr. Charles Kidd[52] says that the following five points are deducible from an increasing experience with chloroform: 1. Dilatation or, rather, engorgement of the right side of the heart, may be calculated on, if the post-mortem be made correctly, and the patient may not have died of simple syncope rather than this apnœa. 2. Chloroform may be recovered by distillation from the blood and lung tissues. 3. Chloroform acts slightly upon the blood corpuscles, they being jagged or crenated at the edges, while ether breaks them up; the lungs, however, are usually anemic or pale under chloroform. 4. Chloroform is more fatal in the struggling stage, or if there be signs of delirium tremens present. 5. Great cau-

[50] Fraenkel, Virchow's Archiv. 1892, Vol. CXXVII. p. 381; Strassman, Berlin, Klink, Feb. 1, 1898.

[51] Blyth, op. cit. p. 150.

[52] Edin. Med. Jour. Sept. 1870.

tion should be observed in believing the confused ideas of women menstruating at the time of taking chloroform.

Dr. Stephen Rogers, president of the Medico-Legal Society of New York, has contributed to the Journal of Psychological Medicine for October, 1871, a very interesting essay on the question "whether chloroform can be used to facilitate robbery." He maintains the negative in an argument from which we extract the following:—

"Referring to the case of the jeweler's shopman, who alleges that the wife covered his face with a handkerchief, while the husband held his hands, it must be obvious to any one at all acquainted with the use of chloroform, that the theory of his allegation is preposterous. Would a strong man, determined to save himself from impending suffocation, stand passively and allow a man and a woman to practise the administration of chloroform on him? He could at least have fallen down and turned his head away enough to have enabled him to scream for help, like the man in Kendal, or the young woman in the stable yard. From all the light which practical experience and the investigation of crimes throw upon this case, there seems little doubt that the shopman was party to the robbery.

"It is at least a very suggestive case, as showing that there is great liability to err on the part of courts in accepting this kind of allegation, unless, upon detailed inquiry into the circumstances and the manner of the alleged giving of chloroform, they be found consistent with the thoroughly well-known facts and phenomena uniformly attending the administration and action of this agent.

"Any inconsistency such an inquiry might develop should be accepted as indicating honest delusion, studied deception, or complicity in crime.

"I feel convinced that such a test would exclude at least nineteen in twenty of all these cases of the alleged felonious use of chloroform and similar agents, as mere fictions.

"But as there may be, as in times past there has been, a popular disposition to accept the statements made by the alleged victims of the felonious use of chloroform, and by their friends, as true, notwithstanding what I may say, or other authors may say to the contrary, with the single motive of bringing before the public and the legal profession all that may be true and well authenticated in this matter, I have challenged, and now repeat it, the production of any proof of the successful use of chloroform on the human subject to facilitate robbery, in a single instance.

"As I have before had occasion to say, when any such proof is fur-

nished me, that robbery has ever been committed by means of the use of chloroform received unconsciously by the person robbed, or given forcibly against the resistance of the person robbed, I will be ready to admit it, and this society will promulgate the fact to the world.

"And I cannot conclude these remarks in a more truthful and forcible manner than by adopting the language of Dr. Snow, who so long ago said: 'The public have been greatly and unnecessarily alarmed about the employment of chloroform by thieves; what they really have to dread is that robbers will still resort to the old means of the bludgeon, the pistol, and the knife, and not to one, which, like chloroform, allows the victim so good an opportunity of escape, and themselves so great a chance of detection.' "

Dr. Snow says: "The sensation of pungency in the nostrils and throat that is caused by this agent, when its vapor is in sufficient quantity to produce any effect on the sensorium, is so strong and peculiar that no person can take a single respiration without being aware that he is inhaling something very unusual. Chloroform, in fact, can never be administered without the consent of the party taking it, unless by main force, which has to be used in the case of children who are not old enough to be reasoned into taking it. If a child be asleep when the process of inhalation is commenced, it nearly always awakes before being made insensible, however gently the vapor may be insinuated. As breathing is perfectly under the control of the will, a person would, on finding such a strange attempt being made upon him in the public street, instantly hold his breath, and use all his powers of resistance to repel the assault,"[53] etc. Lord Campbell, in his speech in the House of Lords, advocating the adoption of the bill making unlawful administration or application of chloroform and other stupefying agents felonious, made the following remarks: A most respectable physician had done him (Lord Campbell) the honor to write him a letter, which had been printed, and which stated that the fear arising from the use of chloroform in this way was altogether imaginary, that no strong man who made resistance could possibly be chloroformed. He believed that was true; but in the case of those who were not strong, and were unable to resist, it might happen to many of that class that the chloroform could be employed most effectively for facilitating robbery. The gentleman, to whose letter he had referred, stated that a person thus attacked might refuse to breathe, and that he might turn away his head. But, suppose a wet handkerchief were put to his nostrils, and held there,

[53] London Medical Gazette, 1850.

the man must breathe, and would thus inhale the particular gas that came from the chloroform. It stood, indeed, on record, that, since the discovery of chloroform, persons had been convicted before the competent courts of using that article for the purpose of robbery. He hoped, therefore, their lordships would be of opinion that those who made such an attempt should not be guilty of a misdemeanor only, as was at present the case; but that any person who tried to commit a robbery by means of chloroform or such like substances, though he did not succeed, if convicted, be held guilty of felony, and be liable to be transported beyond the seas.

We have not seen the evidence brought forward in the cases thus referred to, but we do not doubt that it was fully sufficient to establish the fact of chloroform having been used for the purpose alleged; the only recorded instance which we have met with is the following; its employment was, however, as will be seen, unsuccessful: A gentleman named Mackintosh had retired to bed at a hotel in Kendal. He was awakened about twelve by a man attempting to suffocate him by means of a rag steeped in chloroform. Mr. Mackintosh, who is an elderly man, struggled desperately with his assailant; but, whether from the fumes of the chloroform, or the disadvantage at which he was taken by his midnight assailant, he felt himself fast fainting, when his cries of "help! murder!" roused the house. When the landlord made his way into the room, Mr. Mackintosh was almost powerless, and his assassin or robber was lying upon the bedding, which had fallen upon the floor in the scuffle, apparently sound asleep. On being roughly shaken, the latter professed that he had long been a sleepwalker, and appeared to be astonished to find himself where he was. A policeman was sent for and the man taken into custody. A strong smell of chloroform was perceived by the parties who entered the room, upon the alarm being given, and a bottle containing chloroform was found under Mr. Mackintosh's bed, and a similar bottle in the carpet bag of the prisoner, who had been at the hotel several days. The probability was that the ruffian was secreted under the bed when Mr. M. retired, as the latter had previously placed a chair against the door to prevent intrusion, there being no lock upon the door.[54] This criminal escaped with eighteen months' imprisonment, the offense not being a felony at that time, since there was no intent to commit murder shown.

The following testimony is taken from the records of a trial (New

[54] Med. Gazette, 1850.

Bloomfield, Perry county, Pa., January 18, 1871) for an attempt at robbery by the use of chloroform:—

F. F. Maury, M. D., recalled: Chloroform very, very often produces resistance. It sometimes produces irritation and sometimes a depressing feeling. It produces vomiting. If the stomach is full, nausea and vomiting almost always follow. Sometimes it does not. I experimented with chloroform in six sleeping persons. Out of that number all resisted, more or less. Two men waked up immediately, and one remarked, "You are trying to give me something." Unquestionably, it requires more chloroform to produce death in a recumbent position than in an upright posture. One man cannot administer chloroform to another.

Chloroform will not mix with water; will mix with alcohol. Thrown into water, it breaks into small pieces, as it were. I administered it to a child four days old. It offered resistance. With my experience, I could not administer chloroform to four persons.

B. Howard Rand, M. D., sworn: Am professor of chemistry and lecturer on Medical Jurisprudence in Jefferson Medical College. Cases are numerous where persons have been overcome by smoke and noxious gases, and have perished without attempting to escape. They have been found near limekilns, brickkilns, cementkilns, and in houses on fire. The gases formed when wood or coal is burned are chiefly carbonic oxid and carbonic acid. They are both capable of causing death by suffocation. Carbonic oxid is by far the more poisonous; a single inhalation of the pure gas will produce insensibility; it acts like chloroform to produce insensibility, but is vastly more powerful; death is the rule and recovery the exception, after the inhalation of carbonic oxid, while the reverse is the case with chloroform. When a house is burning, insensibility may result, in part from the diminution of the oxygen within it. When the proportion of oxygen falls below about 18 per cent, insensibility will follow. The vapor of chloroform is 4.2 times heavier than an equal bulk of air. Gases and vapors diffuse or mingle more slowly as they are heavier. Chloroform vapor, formed from the liquid poured on the floor, would form a layer near the floor, from the upper surface of which, supposing the air of the room to be quiet, diffusion would slowly take place, so that the smell of chloroform would be perceptible in the room, but not enough of the vapor would arise to stupefy; the greater part of it would make its way under the door and through cracks, if any existed, in the floor. This vapor is so heavy that it can be poured like a liquid from one vessel into another. It is not

inflammable, and puts out a light. The experiment detailed by Dr. Maury was made at my suggestion, and as a participant I confirm his statements. We concluded the experiment at the end of fifty minutes because we were satisfied that no different result would be arrived at by prolonging it. The only effects noticed were quickening of the pulse of those in the close room, with a pound of chloroform on the floor, except that I was nauseated by the smell of the chloroform vapor, but did not vomit. Mr. Boman remained the whole time lying on the floor with his head resting on his hand, at an average distance of 17 inches from the floor, and was unaffected, except as before stated.

Cross-examined.—Chloroform vapor, as ordinarily administered, when it kills, does so, not by deprivation of oxygen, but from its own noxious qualities. In limekilns a large amount of the noxious gases arises from the decomposition of the limestone, which consists of lime combined with carbonic acid; in the case of brick and cementkilns, the noxious gases arise from the fuel. The proportion of carbonic oxid to carbonic acid in any case will vary; in a smouldering combustion, one where the supply of air is limited, carbonic oxid would predominate; where the supply of air is abundant, carbonic acid would be formed in larger quantity. Anthracite would, weight for weight, yield more carbonic oxid than charcoal, and charcoal than wood. Ordinary smoke contains, besides the noxious gases mentioned, many other bodies, as steam, tar, wood spirit, wood vinegar, etc., the nature and proportions of which will vary with the dryness of the wood and the temperature or degree of heat to which it has been subjected.

Several remarkable instances of robbery of persons designedly rendered insensible by chloroform have lately been reported in the newspapers of this country; although they may be authentic, we do not feel warranted in further alluding to them while unable to attribute them to responsible sources. It is obvious that a person may allege that he has been robbed or maltreated after being rendered insensible by chloroform, and also that the allegation may be false, and be put forward so as to divert suspicion or awaken sympathy.

541. Detection and tests.—Chloroform, boiled with a few drops of a 10 per cent solution of resorcin and a few drops of 10 per cent sodium hydrate, becomes reddish-yellow and shows a green-yellow fluorescence.

A blue color is produced on the addition of a little beta-naphthol in a small quantity of caustic potash solution to the suspected liquid,

and warming if chloroform be present. Chloroform precipitates red cuprous oxid, when boiled with Fehling's solution.

On warming a trace of chloroform with a little anilin and alcoholic potassium hydrate, isobenzonitrite is formed, and may be recognized by its odor. Delicacy 1:5000.

Chemical examination is a very easy matter if not delayed long after death; but unless the chemical research is undertaken very soon, the volatile agent will not be detected.

The method to be employed,[55-6] which is by far the best one, is that proposed by Duroy, and consists in forcing over from a retort, warmed slightly (about 107°F., 42°C.), and containing blood, liver and brain tissue finely divided, the vapor of chloroform, through a hard-glass or porcelain tube heated red hot into a potash-bulb containing a solution of nitrate of silver slightly acidulated with nitric acid; if chloroform be present, a flocculent, white precipitate of chlorid of silver will be seen in the silver solution, and white fumes will be seen issuing from the orifice of the potash-bulb. These fumes are those of chlorin gas, which can easily be recognized by inserting a piece of moistened, blue litmus paper into the exit tube, when the chlorin fumes will first turn the litmus paper red, and finally bleach it. The vapor may be forced out of the retort by a pair of bellows attached to a tube resting just above the organic matter, or else by an aspirator placed at an outlet of the potash-bulb, or, still better, by attaching an ordinary gas holder filled with air to a tube which just dips beneath the surface of the fluid in the retort, and allows the air to be forced slowly through the fluid. Care should be taken to force the vapor through the combustion tube quite slowly. By this process the chloroform is decomposed into chlorin and hydrochloric acid, which forms chlorid of silver with the silver solution. This latter may be recognized in the following ways: It is insoluble in water and boiling nitric acid,[57] but is easily soluble in a slight excess of ammonia; and if the solution containing the precipitate be boiled, black metallic silver will be formed. Provided no white, flocculent precipitate has been produced in the silver solution by the above process in fifteen minutes after the combustion tube has been heated to redness, the experimenter may fairly conclude that no chloroform is present.[58]

[55-6] Du Rôle de l'Alcool et des Anesthétiques dans l'Organisme. Lallemand, Perrin, et Duroy, Paris, 1860.

[57] Cyanid of silver is soluble in the latter, and can thus be distinguished from the chlorid.

[58] *Vide* some experiments concerning chloral, by the editor of the 3d ed. in the New York Med. Jour. for June. 1872. Also, Sansom Chloroform, 1866; Lindsay & Blackiston, p. 45.

Any free hydrochloric acid in the tissue must be tested for, first of all, by litmus paper, and if it is present, a little soda or potash should be added. If chloral had been taken by the patient just previous to death, and the soda and potash be added to the tissues, the chloral would become decomposed into chloroform. These three substances, hydrochloric acid, chloroform, and chloral hydrate, can all be detected by the same process and apparatus, and at the same time. For the detection of the hydrochloric acid, the air should be forced through the mixture in the retort before the combustion tube is heated, when, if hydrochloric acid in the free state is present, a precipitate of the chlorid of silver will appear in the silver solution; if none appears in a few minutes, then the tube should be heated, when, if chloroform is present, the precipitate and fumes mentioned above will be seen; if this result does not take place, then soda or potash should be added to the contents of the retort, when, if chloral is present, a precipitate of chlorid of silver will be formed in the potash-bulb, and the fumes of chlorin will be seen.

XVII. Creasote.

542. In general.—Creasote (kreosote) is a compound consisting largely of guaiacol and cresol. Its use as an application to relieve toothache, being easily accessible, has caused by accident a few cases of fatal poisoning.[59] One of these is reported in Mr. Blyth's table "from accidental poisoning," and other accidental cases are reported; there are also cases of the murdering of infants by creasote.[60] There is another case of an adult woman who died in thirty-six hours after swallowing 2 drams (7.8 c.c.); while another patient, after drinking 6 drams (23.2 c.c) recovered.[61] As creasote is a mixture of crude phenols derived from the distillation of wood tar, its composition is somewhat complicated, but the chief poisonous elements are guaiacol, oxy-cresol, and cresol; by chemical reagents it is difficult to distinguish creasote from cresylic alcohol (cresol).

The constituents of creasote have similar poisonous effects to those of cresylic acid (cresol), which we have before described under the head of carbolic acid (p. 386); guaiacol produces similar poisonous effects to those of the anilin salts and carbolic acid; its application externally of 30 grains (about 2 gms.) has been followed by death.[62]

[59] Manovitz, Ann. d'Hygiène, 3s. 1882, Vol. VII. p. 175.

[60] Puerckhauer, Friedreich's Blaetter, Nuremberg, 1883, Vol. XXXIV. p. 430.

[61] Wochenschr. 1894, p. 219.

[62] Lewin, Lehrb. der Toxikol. 1897, p. 216.

The action of guaiacol, as well as cresol, quinol, catechol, resorcinol, both chemically and physiologically, produces similar symptoms of poisoning to that of phenol (see carbolic acid and its derivatives § 394); the reader is therefore referred to the action described under the heads of carbolic acid and anilin. They are, however, much less poisonous than phenol.

XVIII. Croton Oil.

543. Occurrence and properties.—Croton oil is a fixed oil expressed from the seeds of Croton Tiglium. It is a pale yellow, somewhat viscid, and slightly fluorescent liquid, having a slight fatty odor and a mild oily, afterward acrid and burning, taste. It contains, besides the glycerids of oleic, crotonic, and fatty acids, a peculiar principle called "crotonol," to which the oil owes its vesicating properties. When applied to the skin, it produces a pustular eruption; and when taken internally, is a drastic cathartic. It has an acid reaction to litmus paper, is soluble in alcohol, ether, chloroform, and in the fixed and volatile oils.

544. Poisoning from use of.—Croton oil has been classed as a poison because its medicinal dose is so small that by accident (in one case by design) this may be easily exceeded, results of which, as a strong irritant to the intestinal canal causing excessive purging, may be followed by death. The following cases are recorded of deaths by the accidental swallowing of an overdose: Blyth[63] 1 case only; other cases are recorded in medical literature.[64] One death is also recorded from the swallowing of an overdose in whisky as a practical joke;[65] in this case there was no sign of a purgative action of the drug. In one case strawberries and cherries were filled with croton oil,[66] and thus administered with murderous purpose, or continuous administration of small doses had been given for a long period of time, for the purpose of setting up chronic inflammation of the digestive tract for the same purpose.

545. Symptoms.—Its internal use is followed by intense burning and irritation in the mouth and throat, excessive salivation, and vomiting; these symptoms are succeeded by severe pain in the belly, and by profuse diarrhea accompanied by tenesmus, the discharges sometimes being bloody; the surface of the body is covered with a cold

[63] Op. cit. p. 30.

[64] London Lancet, 1870, Vol. I. p. 553; Adam, Edin. Med. Jour. 1856, Vol. I. p. 932.

[65] Ellis, Am. Jour. Med. Sci. 1874, Vol. LXVII. p. 436.

[66] Mayet and Hallett, Ann. d'Hygiène. Pub. 1871, Vol. XXV. p. 192.

sweat, pulse becomes smaller and irregular, the respiration slow and shallow; cyanosis (a muddy tint to the skin) and the diminution of the body temperature may then follow; finally, delirium, collapse, and death occur from gradual cessation of the respiratory movements. The local action of croton oil on the skin produces burning, redness, and vesication; the vesicles later containing pus. One tenth of a milligram of croton oil on the tongue causes intense burning, which may continue for hours.

546. Lethal dose.—Old specimens of croton oil have a stronger action than fresh or purer samples, on account of the free presence of more crotonoleic acid. As the first effect of the croton oil is to induce severe vomiting, by which much of the dose is lost, it is difficult to tell what amount in one dose should be considered fatal. On an empty stomach its action would, of course, be more intense. One or two drops have been followed by serious symptoms of poisoning, and 20 drops have caused death; a child about a year old died six hours after swallowing a dose of 2½ drops.[67] Recovery has occurred after a dose of 60 drops, and even after 1 ounce (30 c. c.);[68] 2½ teaspoonfuls have been followed by death within four hours. The quarter part of an old seed, which was chewed, was followed by serious symptoms of poisoning.[69]

547. Treatment.—The emetic action of a subcutaneous injection of apomorphin, or a thorough evacuation of the stomach by the stomach pump, should be immediately attempted, after which emollient drinks should be given, followed by morphin to check the reflex action of the mucous membrane of the intestines; to counteract the syncope, medicinal stimulants are required.

548. Action.—Apparently, the action of croton oil is due to its decomposition in the intestines into glycerin and crotonoleic acid. Kobert states that, under the name of crotonolglycerid, this is found in its neutral form in large quantities in the fresh seeds of Croton Tiglium; but that this glycerid is found diminished in commercial croton oil. When introduced under the skin, the neutral crotonolglycerid is inactive; but its "terrific action" is demonstrated when taken into the stomach. In addition to the vomiting and purging action of this irritant drug, he mentions an eczematous eruption on the skin, as a symptom.

549. Post-mortem appearances.—The lining membrane of the whole

[67] Med. Times and Gazette, 1870, Vol. II. p. 466.

[68] Bunting, Boston Med. and Surg. Jour. 1868, Vol. LXXVIII. p. 294.

[69] Schulz, Therap. Monatssch. 1889, Vol. III. p. 89.

gastrointestinal tract, after fatal poisoning by excessive doses of this drug, is found swollen and reddened and detached in places; hemorrhagic spots may be found in the intestines, but in some cases these signs of inflammatory action are not found.

550. **Detection and tests.**—The material under investigation is finely comminuted, and slightly acidified with tartaric acid, then rapidly extracted with ether, filtered, and the filtrate evaporated, when the croton oil, together with other fatty substances, is left in a residue, which should be tested as follows: A little of this residue may be rubbed upon the flexor surface of the arm, observing any eruption that may occur. A small portion may be administered to a cat or dog, the cathartic effect being noted.

There are no distinctive chemical tests that can be used satisfactorily with the small amount that may be present in the stomach or intestinal contents.

XIX. Digitalis.

551. **Properties.**—Digitalin may be obtained in crystalline form, but is usually seen in a form of spherical masses. It is soluble with difficulty in water, even at a boiling temperature; it is readily soluble in alcohol and in a mixture of alcohol and chloroform; it is slightly soluble in absolute ether, pure chloroform, and benzol; benzol will remove it from acid solutions; concentrated hydrochloric acid colors it yellowish-green; concentrated sulphuric acid a greenish-brown, which changes to a beautiful violet-red color if exposed to the action of bromin vapor, and if water is then added, to an emerald-green. Digitalein is soluble in all proportions in water, and is readily soluble in alcohol and ether; it is insoluble in benzol, but slightly soluble in chloroform, which removes it from acid solutions; concentrated hydrochloric acid gives it a light yellow color; concentrated sulphuric acid colors it reddish, which changes to a violet when treated with bromin vapor, and then to a green if diluted with water. The most important test, however, for both digitalin and digitalein, is the physiological test, which is best performed with frogs, since the heart can be exposed, and the action of these principles upon the heart be seen; very small amounts suffice to diminish the number of pulsations, and finally to cause cessation of the heart's action; this cessation taking place during the systole.

552. **Occurrence.**—In 1874, Schmiedeberg discovered in the Digitalis purpurea, or common foxglove, three bodies possessing the char-

acteristic action on the heart, and named them digitalin, digitalein, and digitoxin. These are accompanied by digitonin, one of the saponin series, and by various products of decomposition of the four chief constituents. The action of "digitalis," so called, is more or less the action of all these substances. The digitalins of commerce contain mixtures of these various bodies. In the crude preparations, the solution of these bodies is doubtless facilitated by the presence of saponin substances. These substances have been examined from a chemical point of view more carefully than other plants of this same series, but as yet the results are very unsatisfactory.

553. Preparations.—The leaves of Digitalis purpurea, Linné, collected from plants of the second year's growth; extract of digitalis, fluid extract of digitalis, infusion of digitalis, and tincture of digitalis.

554. Statistics of poisoning.—Husemann notes 45 cases of digitalis poisoning, of which 1 was criminal; 1 was caused by mistaking its leaves for those of borage; 42 were the result of the legitimate medicine used; 33 of these, when too large a dose was given; in 3 the drug was used as a domestic remedy; 2 were the result of wrong reading of prescriptions; and 1 was the use of digitalis in a secret remedy; 22 per cent of the 45 cases were fatal. In the returns of the Massachusetts medical examiners, as shown in our table, there were only 3 cases of fatal poisoning as the result of accident or negligence. Maschka, in his Handbook of Poisons,[70] reports a celebrated case in which digitalis was used for the purpose of murder which he quotes from the publication of Tardieu and Rousin in the Gazette des Hôpitaux, in 1864. As noted above it will be seen that most of the deaths caused from digitalis poisoning are from accident or overdose.

555. Symptoms in general.—The leaves of this plant are the part usually employed, although the seeds also contain a proportion of its active principle. The symptoms produced by digitalis, in a poisonous dose, have some peculiarities by which they may be distinguished from those caused by other poisons enumerated in the class of heart poisons. These are characterized by the remarkable property of causing slowness of the pulse. This symptom, which is quite familiar to physicians, was experimentally produced in nineteen patients in Andral's clinic, the object being to test the efficacy of the pills of "digitalin" recommended by Homolle and Quevenne. In all of them

[70] Op. cit. p. 495, 1882.

the pulse was reduced gradually about 25 beats, after the use of the pills for a period of time, which varied with each patient.[71]

556. **Cumulative effects of digitalis.**—The effects of this poison are cumulative. We do not mean by "cumulative" that digitalis accumulates in the circulation, and thus produces the action of the cumulative form of poisoning by digitalis (see chap. I. p. 41). It is not unfrequently observed that the poisonous action is delayed, as in the often-quoted case of Koehnhorn,[72] in which, after a single dose of 13 grains (0.85 gm.) of powdered digitalis leaves in the form of pills, one daily, no marked symptoms appeared until the eleventh day; these were gastrointestinal disturbance, and the rate of the pulse reduced to 52 beats per minute; he continued taking the pills, and died from paralysis of the heart on the twenty-eighth day. It is not unlikely that the cumulative form of digitalis poisoning is due to the gradual and continuous effect upon the heart tissue, or upon the center of nerve action which controls the action of the heart through the vasomotor system, arresting its functions, thus causing a paralysis of the central organ of circulation. This has not been definitely settled, and the cumulative form of digitalis poisoning is simply used to express the mysterious action of this drug derived from clinical experience. In the case of Koehnhorn, this military recruit took, during the four weeks before his death, a total of 211 grains (about 13.7 gm.). Two of his companions, who suffered temporarily from the same symptoms, left off the use of digitalis before any serious effects were produced.

This poison may remain some time without any obvious effect upon the system, and then display its properties suddenly in a violent manner, and also after being discontinued, its action upon the system does not immediately cease.

As mentioned in the opening chapter (§ 4), digitalis is sometimes tolerated with impunity in certain conditions,—especially that of alcoholic intoxication. Instances where large doses of tincture of digitalis were unattended with serious results were reported by the medical witnesses at a coroner's inquest at Ramsgate, England.[73]

557. **Symptoms of acute poisoning.**—The following is an example of poisoning with the tincture of digitalis, where a teaspoonful was taken in a glass of water. The symptoms did not manifest themselves until five hours after the dose had been taken; then they commenced

[71] Med. Times and Gazette, Aug. 23, 1873, p. 206.

[72] Vierteljahrsschr. f. ger. Med. Vol. XXIV. p. 278.

[73] Med. Times and Gazette, Aug. 23, 1873, p. 206.

with a feeling of nausea, which increased until violent and frequent vomiting took place. There were great precordial distress, intense frontal headache, dimness of vision with dilated pupil, ringing in the ears, cramps in the muscles, very powerful but at the same time irregular and intermittent pulsations of the heart with diminished frequency (44 in the minute), the pulse strong and tense, the respiration sighing, the thirst uncontrollable, retention of urine, sleeplessness, and great debility. The next day, in addition to these symptoms, there was violent delirium, and from that time they continued very gradually to decrease for ten or eleven days. It took some time for the pulse to become normal.[74] A case is recorded by M. Caussé, of a young woman who was pregnant, and who secretly took a large quantity of the expressed juice of digitalis, either to diminish the edema of her limbs, or to produce abortion. The latter effect was produced and death followed in twelve days, probably as much from the want of medical care as from either the abortion or poison.[75] The tincture is often quite inert, or very feeble in medicinal properties; most of the fatal cases of poisoning by digitalis which have occurred, have been either from the leaves in substance or infusion, or from digitalin. A decoction of the leaves, prescribed by a quack in London, to the amount of 6 ounces, proved fatal in twenty-two hours. Dr. Leroux relates a case of poisoning by digitalin which nearly proved fatal.[76] The dose taken amounted to 0.03 gram, which is equal to about half a grain. In another instance, in which 40 granules, equal to about two thirds of a grain, were supposed to have been taken, the patient recovered under the use of emetics and stimulants.[77] The symptoms, however, were not as urgent as in the preceding case. In still another case which recovered, the dose of digitalin taken was also 40 granules, but the symptoms were somewhat different from those enumerated above. The person, who was a female, states that within an hour she was seized with dizziness, debility, vomiting, and cold sweats, and was unable to feel her pulse beat, or to pass her urine; she lay incapable of moving or speaking, her eyes felt strained, and actually projected considerably; she had hallucinations, also continual nausea, extreme tenderness of the epigastrium, and great thirst; and the pulse fell to 46 in the minute.[78]

Poisoning by digitalis is marked by signs of irritation of the stom-

[74] Union Médicale, No. 112. 1851.
[75] Bull. de Thérap. LVI. 101.
[76] Union Médicale, No. 99, 1852.
[77] Dr. Ghereau, Union Méd. Jan. 10, 1854. Quoted in Ed. Monthly Jour. for August, 1854.
[78] Annuaire de Thérap. 1858, p. 102.

ach, such as severe vomiting, with sensations of heat in the head, disturbance of sight, and sometimes vertigo. If the patient is quiet and lying prone, the pulse will be full and strong; but after muscular exertion it becomes rapid and weak. Another sign of its irritant action may be abundant salivation; and on continuation of these symptoms of irritant action on the stomach, signs of muscular weakness and syncope occur with a feeble pulse at the wrist, and a tumultuous beating of the heart. The eyes protrude and are bloodshot, and the pupils are dilated. According to Tardieu,[79] the color of a fire appears blue to the person poisoned by digitalis; after the vomiting has ceased, the pressure over the pit of the stomach causes pain. The tongue is coated with a white envelope and is red at the tip, at other times it is dry and retracted. Breathing is sighing, deep and irregular. Sometimes there is diarrhea of the same character as the vomitus, such as glairy liquids of a greenish color. Vomiting may occur as often as fifty times in the first few hours. Hiccough, and involuntary evacuations accompanied by spasmodic movements, precede the fatal result, which may not occur until the eighth or tenth day, though most usually it occurs within the first two or three days. Tardieu cites one case where the victim lived twelve days. Barth[80] relates one case of an invalid who drank about one and three quarters pints of the tincture of digitalis, and expired in three quarters of an hour, exhausted with repeated vomiting. Twenty grains (1.3 gm.) of the extract of digitalis have caused death in nine days.[81] The above symptoms refer only to acute poisoning.

558. **Symptoms of chronic poisoning.**—Chronic or slow poisoning follows the continued administration of small medicinal doses, in which case, though the medicine has been tolerated for several days or even weeks, violent symptoms of poisoning suddenly appear with syncope, or acute frontal headache with obscuring of vision. In one case Tardieu cites that a paralysis of half of the body followed this syncope, which latter was repeated, accompanied by vomiting and diarrhea, convulsions, delirium, marked slowing and feebleness of the pulse, and death. In these cases of slow or subacute poisoning death may be rapid or may not occur for several days.

Tardieu states that in the thesis of Ducroix,[82] it is cited that two thirds (nineteen) of twenty-four very severe cases of poisoning by

[79] Op. cit. p. 753.

[80] Bull. de la Société Anatomique, p. 685, 1849.

[81] Tardieu, La Clinque, Paris, 1867. p. 685.

[82] De l'Empoisonnement par la digitale, Paris, 1865.

this drug recovered, so that it should be observed that death does not always follow the very severe symptoms above related.

The symptoms of digitalis poisoning are not the same nor as marked by severity in some persons as in others, and are more or less tardy or rapid in development according to the size of the dose taken. The action upon the heart, in reducing the number of pulsations per minute in the pulse, very generally appears, and may persist after the discontinuance of small medicinal doses continuously administered. The acceleration of the pulse is succeeded by the sinking down of the pulse rate to 50 beats per minute or even as low as 25. With the reduction of the pulse rate, nausea, and disturbance of the digestive organs appear, and are followed by disturbances of the nervous system, as above related in the acute form of poisoning, such as ringing in the ears, disturbance of vision, occasional blindness, confusion in the appreciation of colors, and delirium of a mild, quiet nature; later on, the digestive disturbances become more severe, continued with swelling of the tongue and with a fetid breath; the urine is retained; spasmodic hiccoughs and muscular contractions are followed by death.

Cumulative form of poisoning is marked by the sudden appearance of the above symptoms with fainting, and occasional paralysis of one side of the body may supervene. These symptoms may pass into death, without warning. Occasionally, the above symptoms are varied, and the shock to the nervous system is more predominant than the disturbances of the digestion; hallucinations are more marked, and chills, with chattering of the teeth, coldness of the surface, and perspiration, also irregularity of breathing and protrusion of the eyeballs are accompanied with thirst, general malaise and feebleness, absence of diarrhea, and retention of the urine without distended bladder. When the above symptoms abate, there may be a voiding of a red-colored and offensive urine, and an increase in the pulse rate (Ducroix).

559. Lethal dose.—Individual idiosyncrasies, or results of continuous administration of digitalis and its preparations, leave the question of the minimum dose which produces fatal poisoning an open question; 36 grains (2.34 gm.) of the powdered leaves have caused fatal poisoning, while, on the other hand, recovery has followed the ingestion of 1 ounce (30 c. c.) of the tincture of digitalis. When the heart is abnormal, or there is some disturbance in the vasomotor system which controls the heart's action, smaller doses than the above seem to have been followed by severe, and sometimes fatal, poisoning.

The various active principles in commercial use, made from the digitalis leaves, do not all possess the power of producing similar symptoms to those following the ingestion of the leaves or their infusion and tincture; for instance, digitoxin is found in larger quantities in Nativelle's digitalin than in that of Homolle's or in the German digitalin. Digitoxin, according to Kopp's experiments,[83] is from six to ten times stronger than digitalin or digitalein. Digitoxin would probably be fatal to an adult man in the dose of 1/16 of a grain (4 mg.); and digitalin or digitalein in doses of .3 of a grain (18 mg.). The ordinary digitalin has often been taken, without fatal effects, in doses of .13 to .15 of a grain (8 to 10 mg.); while 2 milligrams of this commercial article have been followed by poisonous symptoms in one case. The limit of a dose which might be dangerous to an adult is probably 1/16 of a grain (4 mg.). These doses of the active principle of digitalis found in commerce are given as a single dose, because, if often repeated, as has been before mentioned, serious, if not fatal, symptoms may follow as a chronic poisoning.

The following table is given by Blyth,[84] as showing the maximum quantity of the different preparations of digitalis which can be administered:—

	Single Dose.		Per Day.	
	Grains or minims.	Grams or cubic centimeters.	Grains or minims.	Grams or cubic centimeters.
Powdered leaves	4.5 gr.	.3 gm.	15.4 gr.	1.0 gm.
Infusion	480. min.	28.3 c. c.	1440. min.	84.9 c. c.
Tincture	45. "	3.0 "	135. "	9.0 "
Digitalin	.03 gr.	.002 gm.	.09 gr.	.006 gm.
Extract	3.0 "	.2 "	12.0 "	.8 "

560. Treatment.—There is no known antidote to arrest the poisoning from digitalis.

561. Action.—Digitalis is usually classed as the type of heart poisons. This group of heart poisons has been much extended in recent years, and includes, among other glucosids, antiarin, an arrow poison; helleborin, a glucosid contained in the hellebore family; the poisonous principle of the Nerium oleander; convallamarin from Convallaria; scillitoxin from the squill; and erythrophlein.

The action of this group has its effect on the heart. Stannius experimented on cats; the result of his observations was the irregularity,

[83] Archiv. f. exper. Pathol. u. Pharm. Vol. III. p. 284, 1875.

[84] Op. cit. p. 424.

of the action of the heart, and, finally, its cessation in diastole, from which the heart could not be roused to action by stimulants. Traube[85] experimented on dogs with an extract from digitalis in doses of 7.7 grains (.5 gm.) and 61.72 grains (4 gm.). He noticed in the first stage of this digitalis poisoning that the pulse was frequently retarded, while the arterial blood pressure rose; in the second stage, the pulse frequency, as well as the blood pressure, was abnormally low, but this was not seen when large doses were administered; in the third stage, the arterial pressure was lower and the pulse rate above normal frequency. The retardation of the heart's action is attributable to the stimulation of the inhibitory nerves, and the frequency of the pulse rate noticed later was due to their paralysis. The slow pulse is noticed frequently, even after the section of the vagus nerves, which is explained by the inhibitory action produced upon the cardiac ganglionic center. The vagus nerve is paralyzed, in point of time, before that of the muscular action of the heart. Traube attributed the increased blood pressure to increased energy of the heart's contraction; this, through the motor nerve being stimulated at a later period. He explains the abnormally low pressure as caused by the commencing paralyses.

In addition to these direct actions upon the inhibitory nerve centers and on the ganglions in the heart itself, there is another influence from the action of digitalis upon the vasomotor nerves; this action, witnessed by Ackermann on the mesentery, distinctly showed the contraction of the arteries, and the blood pressure therein to rise after section of the spinal cord.

The view, generally accepted, in regard to this effect upon the increased blood pressure, might be thus stated: "The pressure is due to the prolongation of the systolic stroke of the cardiac pump and to the 'stop-cock' action of the arteries; in other words, there is an increase of force from behind (*vis a tergo*), and an increased resistance in front (*vis a fronte*)." (Blyth.) In addition to this action upon the heart, digitalis, no matter how administered, is usually followed by vomiting. Diuresis occurs, but with a diminished amount of urea; this action is wanting in animals.

Ackermann observed that, during the period of increased blood pressure in the arteries, the surface temperature of the body was increased, but internally it was lowered. From Mr. Blyth's experiments on certain insects,[86] by application of a thin paste of dig-

[85] Ann. d'Chite-Kankenhauses, Vol. II. [86] Op. cit. p. 429.
p. 785.

italis to the head of the common blowfly, the first symptom, after the sign of irritation to the point of application, was the difficulty in walking up a perpendicular glass surface, which increased and resulted in paralysis of the legs, before that of the wings; afterwards, the wings became paralyzed, and the fly, if jerked from its support, fell like a stone. The insect then became dull and motionless, and ultimately died in from ten to twenty-four hours. A dose, insufficient to destroy life, did so, on repetition, at intervals of a couple of hours; he draws the conclusion that digitalins are toxic substances to the muscular substance of even those life forms which do not possess a heart.

562. Diagnosis.—The diagnosis of the poisonous action of digitalis would be recognized by the experienced physician from the peculiar effects upon the heart and circulation, which are above described, associated with the effects upon the digestive tract.

563. Post-mortem appearances.—These appearances are rather vague; the right ventricle and auricle of the heart are usually found filled with blood, the walls of the heart itself being flaccid; the left side of the heart usually is empty; the brain and its membranes are sometimes found anemic; the stomach, and mucous membrane of the intestines, may be in parts ecchymosed, with here and there patches of injection; yet, in many cases of poisoning by digitalis, though the lining membrane of the intestinal tract may show more or less redness, there may be no other appreciable sign of pathological conditions.

564. Digitalin, digitoxin, and digitalein in general.—Digitalin is not the only substance contained in the foxglove, upon which the poisonous action of digitalis depends. There are two other substances of poisonous action; *viz.*, digitoxin and digitalein.

565. Properties of digitalin, digitoxin, and digitalein.—This last is readily soluble in water and alcohol, while the other two alkaloids dissolve well only in boiling alcohol, by some putrefactive process or other decomposition. These three substances split up into two active products called "digitali-resin" and "toxi-resin," neither of which has action upon the heart (Kobert). This observer states that this decomposition product acts like picrotoxin; he also states that digitali-resin may be further split, when it produces the nonpoisonous digitali-genin.

566. Occurrence of digitalin, digitoxin, and digitalein.—Badly dried digitalis leaves, or their infusions, may be decomposed by bacterial life, producing the decomposition of the three above-named heart

poisons; and in using such medicines violent spasms may be caused. The following alkaloids possess some of the poisonous properties of foxglove: Digitalin and digitoxin, the latter of which exists in the leaves of the digitalis plant in the proportion of 1 part to 10,000. Toxi-resin is also intensely poisonous. This is obtained by extracting the dried, exhausted leaves with alcohol of 50 per cent, precipitating this with lead acetate, and washing the precipitate first with a dilute solution of sodium carbonate (to remove coloring matter), and further washings with ether, benzene, and carbon disulphid, in all of which it is insoluble. On decomposing the lead compound, digitoxin may be obtained in colorless scales or needle-shaped crystals.

567. Preparations of digitalin and digitoxin.—Digitalin and digitoxin are not officinal as Pharmocopœia preparations, but may be obtained from certain firms under the name of French or German digitalin, the former under the process of Homolle, and the latter under the process of Walz. Digitalin is officinal in Belgian, French, German, Portuguese, Spanish, and Austrian Pharmacopœias.

568. Detection and tests.—Where the plant itself has been ingested, careful search should always be made in the vomitus and feces, or in the contents of the stomach and intestines, for portions of the plant; the vomitus and feces will usually have a bright green color due to the coloring matter of the plant. This green color can be distinguished from that caused by copper compounds, by its not being changed to an azure-blue color by ammonia water; and from bile, by its not passing through the changes of color characteristic of bile pigment, when treated with concentrated nitric acid. The bright green color is imparted to the contents of the stomach or vomitus by all of the preparations of digitalis; but portions of the plant can be detected only when the powdered leaves, or the infusion or decoction of the leaves, or some other portion of the plant, have been taken. The upper surface of the adult leaf is green, but pale and more or less silvery in the young leaf, and contains very short, transparent hairs, which have a very brilliant and crystalline appearance; the under surface of the leaf is pale and very pubescent, containing a much larger number of the brilliant hairs than the upper surface. The leaves also have another kind of vegetable hair, which is jointed, not crystalline in appearance, and usually made up of two or three segments. Both of these varieties of hair may be easily detected by microscopic examination in the powdered leaf, or in the contents of the stomach, vomitus, etc., when the powder, decoction, or infusion has been taken, except when the last two preparations have been very carefully strained, which is not usually the case.

Both digitalin and digitalein can be isolated from organic mixtures by the process recommended for the opium alkaloids (see § 773), the digitalin being separated by benzol from the acid fluid, and the digitalein by chloroform, and are left as a residue after the evaporation of these fluids.

A body extracted from digitalis by benzol or chloroform gives the following tests:—

It dissolves in concentrated sulphuric acid with a green color, which, on the addition of bromin water, turns purple-red, and on dilution with water, green. When mixed with a mixture of equal parts of concentrated sulphuric acid and alcohol, and heated until it turns yellow, it assumes a blue-green color, on the addition of dilute ferric chlorid. An alcoholic solution of chlorin dissolves digitalin in a yellowish-green color, which, on heating, changes first to violet and then to blackish-green. An aqueous solution of digitalin warmed with a few drops of phospho-molybdic acid develops a fine green color, which is changed to blue by ammonia.

XX. Ergot.

569. Properties.—The chemistry of ergot is still in an unsatisfactory condition, as its active principles seem to be peculiarly unstable and are decomposed by comparatively weak reagents. Kobert has described three bodies as involved in the action of ergot: the first, ergotinic acid, a nitrogenous acid of glucosidal nature; the second, cornutin, an alkaloid; and the third, sphacelinic acid, a resinous acid which contains no nitrogen. Jacobj[86a] has recently stated that the action of the last is due to a very poisonous resinous body, sphacelotoxin.

570. Occurrence.—Ergot is a parasitic fungus (Claviceps purpurea) which grows on the rye (Secale cornutum or cereale) and occasionally on other kinds of grain. It is of interest in toxicology, as the use of bread and meal containing it has frequently given rise to widespread epidemics. Its medicinal effect of stimulating the uterine action and contraction of the blood vessels is well known. If a healthy grain of rye be examined by the microscope, in thin layers, it will be seen to be composed of the seed covering, made up of two layers, beneath which are the gluten cells, while the great bulk of the seed is composed of cells containing starch. In smutty grain (ergotized), dark cells replace the seed covering and the gluten cells, while the large

[86a] Arch. f. Exp. Path. u. Pharm. XXXIX. p. 85.

starch-containing cells are filled with the small cells of the fungus, and numerous drops of oil. Many attempts have been made to isolate the active principle of ergot, but those admitted to contain it, according to Kobert, are cornutin and sphacelic acid (sphacelinic acid). Both of these are recognized as strong poisons; the first produces ergotism upon the involuntary muscles, in the form of clonic convulsions; the second causes, after its absorption at the place of application, local gangrene at the periphery of the body. In Russia and Germany there is used, for subcutaneous administration in midwifery practice, a feebly acid substance, easily soluble in water, as well as in dilute and moderately concentrated alcohol, called "sclerotic" acid. This is active in producing contraction of the involuntary muscles.

Ergot has been known to be produced in bread undergoing fermentation, as can be seen in the following detailed case:—

A remarkable case of fatal poisoning[87] of two persons from eating a bread pudding is reported by Prof. A. H. Allen, of Sheffield, England. Two bread puddings were made from scraps of bread and sandwiches, which had been accumulating for some weeks, together with the addition of milk, eggs, sugar, currants, and nutmegs; these scraps were very moldy, and were mixed with ham, butter, etc. The two puddings were baked in separate ovens, one, a very large pudding, was imperfectly cooked in a notoriously slow oven, and was subsequently eatên by the cook who made it, the proprietor of the eating house, the waiter, a child three years old, and a customer. Every one who ate of this pudding was violently ill with symptoms of irritant poisoning. Only one person ate of the second pudding, but was not made ill. The pulse of the poisoned persons was greatly accelerated; but none had bloody stools. The child and the waiter died, the latter lingering a week. A post-mortem examination of the viscera of the child leading the medical men to suspect its death from poison, the organs, together with the remains of both puddings, were sent to Prof. Allen for a thorough chemical analysis. He made a most exhaustive research, with wholly negative results, and fed a puppy on the suspected pudding for two whole days, without the least appearance of any poisonous effect. The evidence that this suspected pudding had poisoned the persons who ate it was so overwhelming that, at the suggestion of Dr. Tidy, a careful examination for ergot was made by Prof. Allen, as well as a microscopical research for fungi,

[87] Sanitary Record, London, Oct. 25, 1878.

both of which were found. The flour used was examined unsuccessfully for ergot, and this was the case with another pudding made from the same materials used in making the bread. The poisonous pudding showed a large amount of the moldy fungus, but the latter did not give the lake-red reaction with soda, which is characteristic of ergot. The poisonous pudding had no sign of crust on it, but was "sloppy," and of the consistency of bread and milk. The lumps of bread in it were distinguishable, and the starch granules were, in many cases, unaltered.

The verdict of the jury was that the deceased persons "died from eating unwholesome pudding; but how and by what means such pudding became unwholesome, there is no sufficient evidence to show."

According to Dr. Taylor,[88] ergot in large doses causes symptoms of an irritant poison, with diarrhea, and the appearances after death are chiefly patches of inflammatory redness in the mucous membrane of the stomach and small intestine; but, as it is not probable that sufficient evidence of the presence of the active principle of ergot can be obtained from the body by chemical analysis, reliance must be placed on the detection by the microscope of ergot fungus in the matters suspected of furnishing the poison.

Dogs are not readily poisoned by ergot, so that the negative proof offered in the case of Prof. Allen, where the puppy was fed on the poisoned pudding, should have little weight if the poison were the moldy fungus of ergot. Mr. S. A. Wright[89] gave to dogs an amount of ergot equal to 120 grains to each pound of their weight, without injury or poisonous symptom.[90]

571. Symptoms of acute poisoning.—These are twofold: First, the specific action is recognized by the contractions of the involuntary muscles; and second, that of the results of chronic poisoning, to be described later, in the so-called "ergotism," which is so marked by the appearance of gangrene. In acute poisoning the symptoms are mainly vomiting of blood, passing of bloody urine, intense jaundice, and stupor; in other cases, jaundice and vomiting of blood have not been recorded, in place of which there occurs intense gastrointestinal irritation, producing vomiting, colicky pains, and diarrhea. The subacute poisoning may be a secondary result from affections of the nervous system, such as weakness of the limbs, disturbance of vision, retention of urine, delirium, coma, and death.

572. Symptoms of chronic poisoning.—There have been many epi-

[88] Op. cit.
[89] Edin. Med. Jour. 1839.
[90] See Husemann's Handbuch der Toxicologie.

demics of chronic poisoning by ergot, called "ergotism," caused by eating ergotized cereals; and the description of this ergotism dates from the sixteenth century, when a strange, spasmodic, convulsive disease broke out in Hessia; the same disease appeared later in Voigtlander, and in 1702 the whole of Freiberg was attacked. Successive epidemics appeared in Germany and France; in 1710 Dr. Noel had no less than 50 cases under treatment at the Hôtel-Dieu; the mortality was great. Out of 600 cases of ergotism 16 per cent died. During the year 1855, out of 30 with ergotism 23.3 per cent died. The chronic form of poisoning by ergot may be either that of the spasmodic character or the gangrenous form.

The first of these forms of ergotism begins with disturbances of the brain, such as sparks before the eyes, giddiness, noises in the ears, and a creeping feeling about the whole body; there is also loss of sensation in the fingers and toes, and later of the hands and feet, and often of the tongue; diarrhea, vomiting, colic, and other signs of the digestive irritation are usually present; there are tetanic spasms of the muscles, epilepsy, faintings, disturbance of vision, blindness, and amblyopia; skin assumes a yellow color (earthy), and is covered with cold sweat; eruptions and even boils appear; also blebs, or water blisters, like those caused by burns or scalds, have sometimes been seen. Death may occur from exhaustion, in from four to twelve weeks after eating this ergotized grain; even in those who recover, there remain weakness, contractions of groups of muscles, anemia, or disturbances of vision.

Second, the gangrenous form of ergotism is marked by an acute pain in the limbs, or portions where the gangrene is to occur. The tissue in these parts swells with an erysipelatous blush, though there is absence of heat in the part; the gangrene is usually dry, though, sometimes, moist. The extremity below the healthy tissue undergoes a moist process of ulceration, separating this part from the central portion, and, in this way, toes, fingers, legs, and even the nose may be sloughed off. During this process, pyemia, marked by fever, may produce fatal results. In one case which occurred at Wattisham, Suffolk, out of eight in a family, one of whom died (an infant four months old), five persons suffered loss of feet and legs by gangrene separating the healthy from the unhealthy tissue, from the knee down to the ankle. They had been eating freely of wheat which had been cut in the rainy season and had lain on the ground until the grains were black and totally decayed.[91]

[91] Parish Register and Physiosophical Transactions for 1762.

573. Lethal dose.—It should be remarked that small doses of ergot long continued are more apt to produce fatal results of poisoning than a few large doses. It is very difficult to state precisely the minimum fatal dose; 1 fluidram (3.89 c.c.) of the fluid extract caused great sleepiness, swelling and redness of the feet, and formication of the extremities,[92] while ounce doses have been given singly, without causing any poisonous symptoms; death has occurred within one or three days after the first appearances of poisoning.

574. Treatment.—Treatment consists in emptying the stomach and bowels by means of the stomach pump; appropriate medicines, as castor oil, etc., should be administered; if collapse occurs, alternation of warm baths and cold affusions is useful in stimulating the respiratory movements; coffee and tea should be given to act as cardiac stimulants. In chronic cases, where gangrene occurs, the part should be bathed and wrapped in cotton.

575. Action.—The action of ergot has not been definitely determined, but the experiments of Kobert would seem to indicate that cornutin and sphacelinic acid appear to give the physical action of ergot itself. Cornutin, according to Kobert, stimulates the vagus nerve, producing slow pulse and increased blood pressure; afterwards it inhibits the vasomotor center and accelerates the pulse. Tingling of the body and convulsions may follow; paralysis of the extensor muscles, producing deformation of shape of a limb, may result. Cornutin stimulates the uterus to contract, but has not so strong an effect upon the muscles as when given in connection with sphacelinic acid. The latter seems to induce gangrene, which appears in chronic ergotism.

576. Post-mortem appearances.—The post-mortem appearances in animals, on which Kobert experimented, seem to show rapid putrefaction of the tissues, a swollen condition of the mucous membrane of the intestines, and the enlargement of the spleen. He also observed, by the microscope, that the intestinal villi were occupied by a large quantity of micro-organisms in their blood vessels,—especially between the muscular bundles and the deeper layers of the intestines; from this he drew the conclusion that the protective epithelial cells had been destroyed. The mesentery of cats, pigs, and fowls contains numerous small extravasations of blood. The organs in general—especially the subcutaneous cellular tissue—were tinged with biliary coloring matters; from this he inferred that the red blood corpuscles

[92] N. Y. Med. Jour. 1884, XXXIX. p. 68.

had degenerated. He also noticed a hyaline degeneration of the walls of the blood vessels, which showed a quasi-amyloid reaction with iodin. He assumed that the vessels were partly filled with a hyaline mass, in which, later on, a fine black pigment appeared, which would stop up the vessels, and hence cause gangrene.

577. Diagnosis.—In chronic cases of ergotism, by the appearance of the peculiar form of gangrene above described.

578. Detection and tests.—Dissolve 0.5 gram ergotin in an equal weight of water, and, after adding one drop of ammonia, shake out with 1 cubic centimeter ether, evaporate the ether, and dissolve the residue in 1.5 grams acetic acid containing a trace of ferric chlorid. Underlie this in a test tube with a little concentrated sulphuric acid; cornutin gives a bright bluish-violet color at zone of contact. Heat several minutes, with equal bulk ether and a few crystals oxalic acid; ergot develops a reddish color.

As yet it has been found impossible to identify ergot by the isolation of any of its active constituents from animal tissues. Ergot yields trimethylamin, when treated with alkalies; but this is a property common to many organic compounds.

XXI. Jalap and other drastic purgatives.

579. In general.—The chief articles enumerated under this name are jalap, scammony, gamboge, colocynth, croton oil, and elaterium. With the exception of the last two, they are seldom given singly in medical practice, but generally combined with each other and with milder purgatives, or with mercurials, and always in small doses. That they may give rise to fatal consequences from overpurging is not only possible, but is demonstrated by cases upon record. Being the most usual ingredients of quack cathartic medicines,—especially in the pilular form,—they have often been taken in large doses, and have thus caused death by the exhaustion arising from overpurging. One element in the consideration of cases in which death is attributed to the use of any of these drugs should not be forgotten, *viz.*, that the very young, or, on the other hand, the aged, cannot bear the operation of violent purgative medicine, with the same impunity as those of other periods of life; and also that those who are already enfeebled by disease may readily perish from the effects of a comparatively small dose. In general, where the quantity of a drastic purgative taken has been very large, there will be found evidence of inflammatory action in the intestines.

All these irritant purgatives, used as medicines, including another, bryonia, not named above, exert their influence upon the intestinal canal by a peculiar glucosid belonging to each of the above bodies, and very frequently these glucosids form the chief ingredients of quack cathartic pills, which are freely advertised. The compound cathartic pill (U. S. P.) also contains colocynth, scammony, jalap, gamboge, as also aloes, calomel, and cardamom. Occasionally, death is reported to have followed the unadvised use of excessive doses of this group of purgatives, sold under the name of proprietary pills.[93] One case is reported by Taylor, where proprietary pills of this character were used with the intent to commit murder.

580. Symptoms of poisoning; lethal dose.—Dangerous symptoms of overdoses of these substances are, in most cases, vomiting, pain in the belly, violent purging, tenesmus, collapse, and occasionally death. The active principle of two of these substances, jalap resin (jalapin) and podophyllotoxin (glucosid from the North American plant called May apple or mandrake), as also the podophyllin resin (U. S. P.), do not generally produce vomiting, but include the other symptoms above named. Fatal poisoning has also occurred from eating the fruit of the May apple; children have also had severe symptoms of poisoning from drinking the milk of cows that have fed upon the mandrake. Severe conjunctivitis has occurred to workmen powdering the roots and resin.[94] One case of poisoning by podophyllin occurred from taking 5 grains (0.32 gm.) of the glucosid (resin of podophyllum) instead of May apple. Besides the above symptoms named as belonging to this group of purgatives there were facial sweating, slow, weak, and thin pulse, respiratory movements of a sighing character, with difficulty of speech. After a slow improvement and getting up, the original symptoms again returned, with the exception of the purging and vomiting, and twenty-four hours after swallowing the dose the patient became comatose, with a rising temperature and rapid pulse and increased respiration. The smoky urine passed contained albumin and blood cells, and the patient died thirty-one hours after taking the drug. This woman's husband took a similar dose, which was followed by the same set of symptoms; but he recovered after two or three weeks' depressed mental condition.

The fatal dose of this resin of podophyllum, as indicated from these and other cases, would seem to be from 5 to 10 grains (0.32 to 0.65 gm.); yet, in one case 10 grains (0.65 gm.) produced danger-

[93] Paltauf, Wien Med. Presse, 1887, Vol. XXVIII. p. 579.

[94] Webster, Med. Rec. Vol. XII. p. 357.

ous symptoms, from which a woman recovered,[95] while a child recovered after 4 grains (0.26 gm.)

581. Colocynth in general.—Colocynth (bitter apple), Citrullus Colocynthis, contains the glucosid colocynthin, which has not only been used as a cathartic, but is also used as an abortifacient.[96]

582. Symptoms from ingestion of colocynth.—The symptoms following the ingestion of colocynth, while resembling those of the above-named purgatives, have, at times, been followed by an irritation of the kidneys, and when fatal, are marked by delirium, prostration, an irregular pulse, and collapse. The fatal dose of the powdered colocynth fruit would appear to be something under 2 teaspoonfuls. One case, especially, was followed by death after a dose of one teaspoonful; yet, 2 or 3 drams (7.8 or 11.7 gm.) have been taken without fatal results.

583. Gamboge.—Gamboge (U. S. P.), a gum resin obtained from Garcinia Hanburyi, Hooker filius, in the dose of 1 dram (3.9 gm.), as a medicinal purgative, has been followed by fatal results; it is also used as a pigment; it has produced symptoms of poisoning in children who have moistened their lips with brushes upon which the pigment was smeared.

584. Elaterium.—Elaterium (squirting cucumber, wild cucumber), being a drug of such uncertain strength, is not, as a powder, officinal (U. S. P.). Its active principle, elaterin, officinal (U. S. P.), is used in doses of 1/16 of a grain (.004 gm.), repeated every one or two hours to produce watery discharges from the intestines, and for the relief of cerebral symptoms dependent upon diseases of the kidneys. Occasionally, deaths have resulted from excessive doses of this drug. The fatal dose is usually placed at 7 or 8 grains (0.455 or 0.52 gm.), and in cases of feebleness or old age, even less than a grain (0.065 gm.) has been followed by fatal poisoning.[97]

585. Treatment.—Treatment of all of this group of purgative poisons would be the same as that which we mentioned under the head of croton oil; *viz.*, the poison to be removed from the stomach by emetics or the stomach pump, and emollient drinks to be afterwards administered; precaution as to diet should also be strictly enjoined, and in the case of a feeble heart action, a cardiac stimulant, such as carbonate of ammonia, should be administered.

586. Post-mortem appearances.—Besides the local inflammatory ac-

[95] Phila. Med. Times, 1882, Vol. XII. p. 520.

[96] Pharm. Jour. and Trans. 3s. 1878, Vol. 8, Pt. 2, p. 1035.

[97] Am. Jour. Pharm. 1868, p. 373.

tion of the intestinal tract there may be an ulceration throughout its length, and the irritation of the mucous membrane may be extended even into the kidneys and bladder, and there may be congestion in the peritoneum, liver, and spleen.

XXII. Poison ivy; poison oak; primrose; and moccasin flower.

587. In general.—Poison ivy, poison oak, primrose, the moccasin flower (lady's slipper),—all this class of plants may produce, by local contact with the skin, a peculiar form of eruption characterized by itching, irritation, swelling, vesication, and, finally, desquamation of the integument; though poisoning usually results from direct contact of the plant with the skin. Even contact with articles which have touched it, or people who have handled the plant, may convey the poison. The poison is due to the pollen, which contains the oil, and also to the minute hairs which are so abundant over the plant itself;[98] and these are responsible in many cases, where poisoning has occurred by secondhand, and in their direct contact with the plant itself.[99] The active principle of these plants has been shown to be a nonvolatile oil, toxicodendrol: 1/64000 of a grain (1/1000 of a mg.) is sufficient to produce distinct effects, when brought in contact with the skin of some persons. In the trial term of the supreme court of Brooklyn, May 6, 1897, a judgment for $3,500.00 was rendered against the directors of a cemetery, in a case of poisoning shown to have come from contact with poison ivy. The plaintiff was ill for eighteen months.

588. Treatment.—The treatment for the local poisoning from poison ivy is the application of an alcoholic solution of acetate of lead; the alcohol dissolving the poison, and the lead forming an insoluble compound with it. The use of oils is especially contraindicated; for the toxicodendrol is soluble in fatty substances, and will simply spread the poison over the body.

589. Swallowing of berries of poison ivy.—The swallowing of the berries of the poison ivy by children has occasionally happened. The symptoms are those of drowsiness, stupor, and mild delirium;[100] also a rash resembling that of measles, accompanied with itching, suffusion of the eyes, and pain in the throat and stomach, has illustrated the action of the poison when swallowed. Pfaff found, by

[98] Schwalbe, Munich Med. Wochenschr. 1902, Vol. XLIX. p. 1616.

[99] Pfaff, Jour. Exper. Med. 1897, Vol. II. p. 181.

[100] Am. Jour. Med. Sci. 1867, p. 56; Med. and Surg. 1867, Vol. XVII. p. 373.

experimenting on rabbits, that the internal application of toxicodendrol caused inflammation of the kidneys (nephritis).

590. Other plants exhibiting the same local irritation.—Other plants exhibiting the same local irritation upon the skin to that of poison ivy (Rhus toxicodendron) may be mentioned; as certain varieties of buttercups, crowfoot, some species of Euphorbia (corollata[1] and marginata[2]) and Primula obconica.[3]

XXIII. Picrotoxin.

591. Properties.—It is very slightly soluble in cold water, quite so in hot water, is easily soluble in alcohol, difficultly soluble in ether, chloroform, amyl alcohol, and ligroin. It forms colorless crystals, with a melting point of about 195°, which are odorless, but intensely bitter. It reduces Fehling's solution on warming.

592. Occurrence.—Picrotoxin is a bitter, active principle obtained from Cocculus indicus (Indian berry, Levant nut) found in the East Indies. The powdered berries have been used as fish and bird poisons, in medicine chiefly as a parasitic remedy, and as a household remedy for vermin.

This is the fruit of the Anamirta Cocculus. The kernel, which is the only poisonous portion of the berry, has no smell, but an intensely bitter taste. It contains an active principle called "picrotoxin," which is an exceedingly active poison.

Cocculus indicus is chiefly used for the purpose of taking fish and of sophisticating malt liquor. It is also used for the destruction of lice. Several fatal accidents have occurred in this country from it. Dr. Thompson reports one case from its external application. A child aged six years, whose head, after the hair had been cut close, was washed with an alcoholic tincture of Cocculus indicus, was seized, in less than half an hour after its application, with tetanic convulsions. The pupils, during the spasm, were exceedingly contracted, and in the interval between the attacks were dilated to the fullest extent. By touching the eyelids, the spasm could be produced

[1] Bull. Torrey Bot. Club, 1895, Vol. XXII. p. 393.

[2] Jackson Med. Rec. 1897, Vol. LI. p. 636; Schenck. Gaz. 1890, Vol. XV. p. 277. Poisoning may also result from the eating of honey made by bees from flowers of the Euphorbia marginata; the chief symptoms of which are vomiting and purging.

[3] See Bull. Pharm. 1898, Vol. XII. p. 80; Cooper, Med. Jour. 1899, Vol. I. p. 1543; Dreyers, Munich, Med. Wochenschr. 1902, Vol. XLIX. p. 574. A large number of cases are cited in the last article.

at pleasure. The case was treated with energy, but the child died in a few hours. On post-mortem examination, no changes of any note were observed. A younger sister of the deceased, who had also been submitted to the same cleansing process, was likewise attacked in a similar manner. Under the use of counterirritation by mustard, and injections of the tincture of asafetida, she recovered, the convulsions gradually subsiding about three hours after the attack commenced. The next morning a scarlatinous eruption appeared upon the body and arms, which gradually faded during the day.[4]

The following account of several cases of poisoning by the internal use of this substance has been kindly communicated by Dr. Fish, formerly assistant resident physician at the Philadelphia Hospital, Blockley. A strong decoction of this berry (two ounces to a pint of water) is used in that institution for the destruction of vermin upon the paupers. The vessel containing it was, unfortunately, placed near some tonic infusions in use by several patients. Through the ignorance of the nurse, a wineglassful of this decoction was given to each of three persons, and two tablespoonfuls to three others, by mistake for their usual medicine. Two of those who took the largest quantity were seized with convulsions about twenty minutes after they had taken the poison, and died in about half an hour. This happened in the evening, and their muscles were still contracted the next morning. Both of these men were much reduced by intemperance and disease. The remaining four, who were seized within a few moments of each other, and within half an hour after they had taken the poison, presented the following symptoms: Faintness, mental confusion, giddiness, dimness of vision, nausea, excessive thirst, severe pain in the abdomen, and, in one case, insensibility. The pulse was much weakened, and the respiration was slow and labored. Emetics were given to them, and, after the vomiting, mucilaginous drinks and stimulants. They all recovered, but suffered greatly from headache during the rest of the day.

Another case is mentioned in Traill's Outlines; and one is referred to by Taylor, in which the post-mortem examination distinctly revealed traces of gastroenteritis, due to the irritant action of the poison. In this case, however, the patient lived until the nineteenth day.

The following case presents a curious question in the administration of poisons. The prisoner was indicted for administering poison,

[4] Phila. Med. Ex. April, 1852, reported by Wm. B. Thompson, Senior House Surgeon, Emigrants' Hospital, Ward's Island.

and it was proved that two Cocculus indicus berries had been given to a child nine weeks old. The child, after having swallowed them, threw up one by vomiting, and the other passed through her body in the natural course of digestion, and was found next day in her clothes.

Two medical witnesses, called on the part of the prosecution, proved that the Cocculus indicus berry is classed with the narcotic poisons; that the poison consists in the presence of an alkaloid, which is extracted from the kernel; that all the noxious properties are in the kernel; that it has a very hard exterior or pod, to break which much force is required.

One of the witnesses added that the berry, if the pod is broken, is calculated to produce death in an adult human subject, though he did not know how many berries would be required for the purpose; that he thought the poison contained in the kernel of two berries, if the pods were burst, and if retained on the stomach, might produce death in a child nine weeks old; but that the berry could not be digested by the child, and that it would pass through its body, without the pod being burst, and so would be innocuous (as had, in fact, happened in the present case).

The counsel for the prisoner objected that the berries were not poisonous within the meaning of the statute; for though the kernel of the berries contained poison, yet the pod rendered the poison innocuous. The judge (Vaughan Williams) overruled the objection, and left the whole case to the jury. Verdict, guilty.

Judgment of death was recorded, but execution was stayed in order to submit the point raised by the prisoner's counsel to the consideration of the judges. The discussion before them in the Exchequer Chamber is given in detail by the reporter, and is quite interesting.

The counsel for the prisoner observed that the indictment was founded on the statute 1 Vict. chap. 85, § 2, which makes it a capital felony to administer to, or cause to be taken by, any person, "any poison or other destructive thing," with intent to commit murder. The real question is, whether the berries, in the state in which they were administered, were "poison." The prisoner thought he was giving a destructive thing, but did not do so. It was inquired of the counsel by the judges what he would say if arsenic were given in a globule of glass? Again, if arsenic were put in a paper envelope, and that wrapped in oil paper and administered? He contended that in "such states it could not be a destructive thing." But, it was replied, if a person gives poison in too small a dose, you would say that it was

not within the statute, as it could not be destructive. If you are right in so saying, persons might give doses of arsenic, and speculate on the size of the dose. Finally, Chief Justice Wild remarked, "The question here is whether the prisoner administered poison with intent to murder. The kernel of the berry was a poison, but he administered it in a condition in which it was not capable of doing injury. Is that administering poison? If a person administers poison with intent to murder, but accompanies it with something which prevents it acting, we all think it is the offense provided for by this enactment, and that the conviction must be affirmed." Justice Alderson said, "This is very different from the case of a person administering an innocent thing and thinking it poison; there he does not administer poison at all; here he does." The other judges concurred in affirming the conviction.[5]

In addition to picrotoxin there are two other definite crystalline principles contained in Cocculus Menispermum, called "menispermin" and "paramenispermin" (nitrogen containing bases). Picrotoxin appears to possess some of the characters of an acid, and exists to the extent of 1 per cent in Cocculus indicus.

593. Symptoms of acute poisoning.—After the poison is swallowed, there is a burning pain in the gullet and stomach, followed shortly by salivation, nausea, vomiting, and diarrhea. There is also cerebral disturbance, as shown by confusion of mental action, dizziness, and drowsiness; with these symptoms, coldness, profuse sweating, and unconsciousness appear. The face is pale, the pupils are alternately contracted and dilated; respiration is hurried, and later, the movements become slow; convulsions begin, as described in the cases above quoted, in about twenty minutes. In a case reported by Sozinskey,[6] these powerful, general convulsions followed each other every five minutes, each lasting about two minutes; between the convulsive movements there was perfect relaxation; opisthotonos was very marked. At the beginning of each attack there was an outcry, as in epilepsy, and frothing at the mouth. In Thompson's case, pupils were contracted during the convulsion and dilated during relaxation; touching the eyelids caused a spasm, showing the increased reflex excitability similar to strychnism, in which respect these convulsions are similar. If the patient survives the convulsive part of

[5] Am. Jour. Med. Sci. April, 1851. from *Reg.* v. *Clanderoy*, Carrington and Kirwan's Nisi Prius Reports, Vol. II. p. 709.

[6] Shaw Med. News, 1891, Vol. LIX. p. 38.

the attack, he may die from inflammation of the intestines several days later.

594. Detection and tests.—A green color develops on adding a solution of potassium bichromate to picrotoxin. A fragment of picrotoxin, added to two grams of nitric acid (sp. gr. 1.4) and warmed, gives a yellowish-red mass, which turns to violet-red on the addition of potassium hydrate solution, and, on warming, changes to the color of old blood.

A minute quantity of picrotoxin, evaporated to dryness with concentrated nitric acid, and the residue moistened with strong sulphuric acid, on the addition of potassium hydrate, gives a brick-red color.[6a]

The method proposed by Palm[6b] is as follows: Evaporate the material to dryness and extract with acidified water. Extract this solution with ether, filter, evaporate the ether, take up again with water, and decolorize with animal charcoal. Then add neutral lead acetate to complete precipitation, filter, and to the filtrate add freshly precipitated lead hydrate, which precipitates the picrotoxin as a lead compound. This lead compound is then suspended in water, decomposed by H_2S, and the picrotoxin extracted from the aqueous solution with ether.

Another method of isolating picrotoxin from organic mixtures is the ordinary method for alkaloids, the picrotoxin being removed from the acid solution by chloroform.

The best method for the separation and detection of picrotoxin in beer or ale is that recommended by W. Schmidt, slightly modified by Dragendorff.[7] This method depends upon the principle that the picrotoxin is not precipitated by basic acetate of lead, and also that it can be removed from its solution in acidulated water by shaking with amyl alcohol or ether. The beer or ale is first evaporated to the consistence of a syrup, then a sufficient amount of warm water added to render it fluid, and mixed with animal charcoal (5 or 6 grams for a bottle of beer). After several hours it is filtered, the charcoal washed, and the filtrate treated with basic acetate of lead in excess, filtered, the excess of lead in the filtrate separated by passing a current of sulphureted hydrogen gas through it; the sulphid of lead thus formed will carry down a number of impurities; filter from

6a Langley, Zeitschr. f. anal. Chem. II. p. 404; Cheopinsky, Jahresber. d. Chem. 1884, p. 1644.

6b Zeitschr. f. anal. Ch. XXII. p. 274, XXIV. p. 556, XXVII. p. 99.

7 Pharm. Zeitsch. f. Russl. I. pp. 304 and 414.

the sulphid of lead and shake the filtrate, which should not be more than one third of the volume of the original fluid after allowing the sulphureted hydrogen to escape, with five or ten per cent of amyl alcohol; after this has separated, it should be decanted and the shaking repeated with a new portion of amyl alcohol; the mixed amyl alcohol fluids should be evaporated by gentle heat and the residue extracted with dilute alcohol (50 per cent), filtered, and the filtrate evaporated; this residue is dissolved in boiling water to which a few drops of sulphuric acid have been added, the solution decolorized with animal charcoal, filtered, and the cooled filtrate shaken with ether, which will remove the picrotoxin and leave it, after evaporation, as a residue. The picrotoxin can be purified by crystallizing it alternately from its solutions in water and alcohol. The sulphid of lead and the animal charcoal left upon the filter paper in the above process may retain a little of the picrotoxin, and should be washed, dried, and extracted with ether. By this method Schmidt has obtained, on one occasion, four decigrams (more than 6 grains) of picrotoxin from a bottle of beer.

XXIV. Pyridin.

595. In general.—Pyridin, in commerce, occurs as a colorless, volatile fluid having a characteristic odor and taste, and is sometimes used with alcohol to make the latter undrinkable. One case[8] is recorded, where a man, twenty-nine years of age, employed as a stillman in a factory for making tar, accidently swallowed a cupful of pyridin. The symptoms observed were nausea and vomiting; and, when visited by a physician seven and one half hours after swallowing the pyridin, his countenance was pale, and the skin of the lips slightly cyanosed; the tongue had a dry, white coating, and the skin was covered with cold sweat; his temperature was 103.4 F.; pulse 128, weak and intermittent, and the respiration 40 per minute; his breathing was noisy, due to obstruction in the bronchial tubes or from mucous râles; he complained of a sensation of tightness and oppression in breathing, a choking sensation, with pain down the center of the chest, and pain over the pit of the stomach. Emollient drinks were given, and brandy by enema. He coughed up white and frothy mucus, smelling strongly of pyridin, and his exhalations contained a similar odor. The second day his temperature was 104, but gradually subsided during the day; the expectorations became purulent.

[8] Brit. Med. Jour. 1893.

and the lungs gave physical signs of acute congestion and bronchitis. In a few hours later the temperature of the body rose to 105.8 F.; the patient became delirious, and died forty-three hours after the accident.

596. Post-mortem appearances.—The larynx, trachea, and bronchi were coated with a friable, yellow membrane; the bronchi contained purulent matter; the lungs were congested and edematous. There were, also, congestion in the lining membrane of the œsophagus and at the cardiac portion of the stomach, and less marked, similar appearances at the pyloric orifice and the upper portion of the duodenum.

Pyridin, according to both Kobert and Blyth, is a component derived from the smoke of tobacco, and the reader is referred to the article on nicotin for further information.

XXV. Resorcin.

597. In general.—Resorcin (resorcinol and metadioxybenzol, U. S. P.) is described as a diatomic phenol, which should be kept in dark amber-colored vials. It is produced from galbanum and asafetida by treating these resins with potassa; it resembles orcin, the coloring made of orchil. It is closely related to phenol, and is, like it, a powerful antiseptic when diluted with alcohol to 1 per cent. It is much less poisonous than phenol; and the poisonous symptoms following its use in very large doses rapidly disappear, unless the dose is large enough to be fatal, which is found to be, in the lower animals, nearly 1 per cent per kilo weight.

598. Symptoms of poisoning.—In quantities of one third to more than a half of that as above mentioned, these symptoms appear: Trembling; clonic convulsions; and increased movements of respiration and circulation, which last for about an hour. In man the additional symptoms are excitement and vertigo. Clinical experience shows a doubt as to whether the temperature is increased or lowered; if the temperature is lowered, it is but transitory. Resorcin has been used as a medicine in fevers, like many other antiseptics, without producing any remarkable effects. Its internal use has the advantage over carbolic acid, of producing less toxic properties. Its external use in surgery is preferable to carbolic acid, both on account of the absence of the odor, the danger of poisoning, and its solubility

in any proportion of water. The medicinal dose is from 15 to 60 grains (1 to 4 gm.) *per diem,* in cases of typhoid fever and acute rheumatism.

The subjective symptoms are further giddiness, insensibility, cyanosis, and a cold, clammy surface; convulsions rarely occur; the urine has an olive-green color.

Cresol, quinol, catechol, resorcinol, and guaiacol, though chemically and physically similar to phenol, have much less poisonous action than phenol.

XXVI. Salol.

599. Properties.—Salol, phenyl salicylate, $C_6H_4OHCOOC_6H_5$, is a white, crystalline powder, almost insoluble in water, having a faint aromatic odor and a slightly salty taste. It is soluble in alcohol, ether, and benzol. It is decomposed by the pancreatic juice into phenol and salicylic acid.

600. Symptoms.—The symptoms of poisoning, which have occurred in a few cases, will resemble those of carbolic acid and those of salicylic acid poisoning; *viz.,* nausea, vomiting, increase of temperature, drowsiness, bloody urine, and sensation of tightness of the head with ringing in the ears, etc. As in the case of resorcin, the urine voided has an olive-green color. Its poisonous action is more violent when given to persons suffering from nephritis or other disturbance of the kidneys, such as abnormal contraction. One case of this character is reported by Hesselbach, and another case by Sahli.[9] In the first case, the woman had a contracted kidney, and was sick with acute rheumatism; she died on the fifth day after taking 2 drams (7.76 gm.) during eight hours. The second case was attributed to 15 grains (1 gm.) of salol, twelve days after its administration.

601. Detection and tests.—Salol, on heating with zinc chlorid, decomposes into phenol and a salicylate, and the vapors evolved color paper, moistened with ferric chlorid solution, a dirty green, changed to violet with ammonia. Both phenol and salicylic acid can be found in the urine. The material under investigation can be saponified, and then tests made for both phenol and salicylic acid.

[9] Forschr. der Med. 1890, p. 453; see Sahli, *ibid.* p. 661.

XXVII. Santonin.

602. In general.—Santonin is the anhydrid of santoninic acid. It is contained in santonica or Levant wormseed.

Santonica (Levant wormseed), the unexpanded flower heads of Artemisia pauciflora (Weber) gives a neutral principle called santonin (U. S. P.). Santonin is insoluble in cold water, soluble in 40 parts of alcohol at 15° C. (59° F.), in 250 parts of boiling water, and in 3 parts of boiling alcohol; also soluble in 140 parts of ether, in 4 parts of chloroform, and in solutions of caustic alkalies. Santonin troches (U. S. P.) contain 3 grams of santonin, in fine powder, in 100 troches. As santonin is decomposed by light, it should be kept in dark amber-colored vials. Santonin is a glucosid, and is employed as an anthelmintic for roundworms and threadworms. It is usually given to children, in whom more cases of poisoning with santonin have occurred than with adults. The larger part of santonin administered passes through the intestinal tract without being absorbed; but under certain conditions, not particularly understood, much of the santonin is absorbed, and the general symptoms of poisoning occur as follows: Troubles of the vision, such as "yellow sight," sometimes, also, blue.[10] Though these symptoms rarely continue more than twenty-four hours, yet one case is reported in which complete blindness lasted for a week. There may also be disturbances in the sense of taste, of smell, and of hearing. The absorption of larger doses produces disturbances of the central nervous system, which may begin within a few minutes after swallowing the drug. Other symptoms are headache, giddiness, slight clonic convulsions, with disturbance of the mental faculties. These symptoms may be associated with pain over the region of the stomach, vomiting, and labored respiration. After large doses, the convulsions may be succeeded by stupor, loss of consciousness, and death from collapse. The urine voided is colored a saffron-yellow. There may also be clammy skin, edematous eruption of the skin with fever, and symptoms of irritation of the bladder, sometimes bloody urine, and albuminuria. As the excretion is mainly through the urinary system, and this elimination produces local irritation in the uriniferous tubes of the kidneys, the elimination may be delayed, and, in consequence, the action of santonin may produce what is called "cumulative."

[10] Rey, Therap. Monatssch. 1899, Vol. III. p. 532. See, also, Rose, Virchow's Archiv. Vol. XVI. p. 233; Vol. XVIII. p. 15; Vol. XIX. p. 532; Vol. XX. p. 245; Vol. XXVIII. p. 30.

603. Lethal dose.—A fatal dose to a child between five and six years may be 2 grains (13 cg.), and death may follow in twelve hours after its ingestion; yet, when the elimination by means of the kidneys is rapidly performed, a larger dose may be taken with impunity; for instance, recovery in a child has followed the dose of 10 grains (0.65 gm.); a child of five years died in thirty-five minutes after taking 6 grains (0.39 gm.);[11] an adult has recovered after taking 1 ounce of santonin (31.10 gm.) in mistake for Epsom salts. It would seem, therefore, for children under six years a safe medicinal dose should be less than 2 grains (about 13 cg.).

604. Treatment.—Treatment should consist of thorough evacuation of stomach and bowels, and diuretics to assist in the elimination of the drug through the urine. In case of collapse, warm baths alternated with cold affusions should be given. The convulsions should be counteracted by bromid of potassium and chloral.

605. Action.—Experiments on animals seem to show that at first there is an excitation upon the nervous centers, which is soon followed by decrease of excitability. The medulla oblongata becomes affected later; this is the center of innervation for the heart and respiratory movements.

The peculiar action of santonin in exciting the blue and yellow portions of the retina resulting in "yellow sight" was first observed by Hufeland in 1806. If the eyelids are closed while yellow sight is present, the whole field of vision appears violet. De Martigni[12] reports that he found that small doses produced the yellow sight, which might intermit and pass into other colors when larger doses were given. To some individuals it would show the objects red; an hour later, orange, and then again yellow. In another person the effect of the drug was green vision; and in the third case, blue.

606. Post-mortem appearances.—These are not characteristic.

607. Detection and tests.—Santonin, dissolved in H_2SO_4, without heat, and very dilute solution of ferric chlorid added in small, successive quantities, develops a red to purple or violet color, on shaking.

On heating a fragment of santonin with an alcoholic solution of ethyl nitrate, and then adding a few drops of potassium hydrate to the hot fluid, a violet color develops. The reaction will readily detect 0.001 gram.

The material to be examined is made slightly alkaline with sodic hydrate, and then dialyzed to a thin syrup, and kept at about 30°

[11] Kilner's St. Thomas' Hosp. Rep. N. S. 188, Vol. X. p. 246.

[12] Gaz. Hôp. 1860.

with three times its volume of alcohol for twenty-four hours. The mass is then filtered, evaporated till alcohol has disappeared, and the aqueous alkaline fluid is shaken out with benzol. When all the impurities have been removed that it is possible to remove, the solution is acidified with hydrochloric acid and again shaken out with benzol or chloroform. The chloroform or benzol extracted on evaporation will leave the santonin in solution. It can then be tested for by the above tests.

XXVIII. Strophanthus.

608. Occurrence and preparations.—Strophanthus (U. S. P.); the seed of Strophanthus hispidus De Candolle, the pride of its long awn. Tincture of strophanthus, 50 parts of powdered strophanthus seeds in 1,000 cubic centimeters of alcohol and water.

609. Symptoms and action.—Strophanthin, its alkaloid, is a cardiac poison, and the symptoms following its use are similar in most respects to those described under the head of "digitalis;" in other words, it slows the action of the heart, and in large doses produces heart paralysis, with the permanence of the ventricular systole. Dr. Fraser[13] found that the pulmonary respiration continued in cold-blooded animals many minutes after the heart was paralyzed. From his experiments he concluded that the striped muscles of the body are also affected, as shown by the twitching of groups of muscles; that their tonicity is exaggerated and finally exhausted, the functional activity having ceased. He assumed that this change is due to the action on the muscular fiber itself, independent of that of the heart, and also independent of the cerebro-spinal system. He concluded that the reflex action of the spinal cord is suspended after paralysis of the heart; but the motor conductivity of the spinal cord and of the nerve trunks continues after the striped muscles of the body are paralyzed. The lymph hearts of the frog continue to contract for many minutes after the blood heart has been paralyzed. It should be remarked that the course of action of strophanthin is more rapid than that of digitalis.

610. Properties, isolation, and tests.—Strophanthin is a glucosid, the active principle of Strophanthus hispidus. It occurs in white crystalline plates, slightly soluble in water, with a bitter taste, quite soluble in ethyl or amyl alcohol, insoluble in ether or benzol. The

[13] Jour. Anat. and Phys.; Fraser, Trans. Royal Soc. Edinburgh, 1890–91; and Klin.-Wochenschr. 1888, Vol. XXV., p. 115.

strength of the derivative glucosid, strophanthin, varies, as it is liable to decomposition; while if freshly prepared, a slightly increased dose may occasion unusual and severe action upon the heart and systemic blood circulation.

It is extracted from the acid aqueous extract by shaking with amyl alcohol, as described under morphin.

Strophanthin with sodium nitroprussiate and ammonium hydrate gives a red color.

With a drop of ferric chlorid solution, strophanthin gives, on the addition of sulphuric acid, a red-brown precipitate, which gradually becomes green.

Sulphuric acid of about 75 per cent. produces a violet color with strophanthin.

The physiological test is shown by the arrest of a frog's heart in systole.

XXIX. Saponin.

611. Occurrence.—Saponin occurs in soap tree bark and other plants. It is soluble in water and alcohol. Its solution behaves like soap solutions. It is poisonous, but is sometimes added to soda water and beer to produce a permanent froth. In materia medica saponin is used to include the less active glucosids and certain innocuous isomers of the sapotoxins.

Quillaia Saponaria, Agrostemma Githago (corn cockle), contains the glucosid called "saponin" and "sapotoxin." Saponin in the proportion of 1 milligram to 1 cubic centimeter of liquid is very soluble in water, to which it gives a peculiar frothy look like soap solution. It is sometimes used to produce a bead in beer. Its reaction is neutral. It has no odor; but it causes sneezing if applied to the mucous membrane of the nose. The first taste to the mouth is sweet and then sharp and acrid.

612. Action.—The effect on frogs, as shown by Pelikan,[14] when 1 or 2 drops of a saturated, watery solution of saponin were injected under the skin of the leg, caused, in from five to six minutes, great weakness, accompanied, apparently, with loss of sensation. Strong mechanical, chemical, or electrical stimulant applied to the foot excited reflex action, showing that the function of the nerves in conveying nerve influence was still retained. The poisoned muscles had lost their excitability, which ceased just before the period of death.

[14] Berlin klin. Wochenschr. 36, 186.

A concentrated solution applied to the heart of a frog soon arrests its beats; weaker doses first excite and then retard the cardiac action.[15] Blyth, in experimenting with kittens, insects, and infusoria, with small doses, 1/5 to ½ grain (13 to 32 mg.), noticed immediately signs of local pain when they were injected under the loose skin of the back of the neck of a kitten. Five or ten minutes later, respiration was notably quickened, and the animal became lethargic with signs of muscular weakness; just before death, breathing became very rapid with signs of asphyxia.

The action on man has not been thoroughly studied. Doses from 1.5 to 3 grains (.1 to .2 gm.) seem to produce distinct physiological effects, such as increased mucous secretion followed by nausea. It may be assumed, from the foregoing fact, that 40 grains (2.6 gm.) administered subcutaneously to an adult will endanger life. Kobert gives the action of sapotoxin, contained in saponin, as producing bloody urine, inflammation of intestines, disassociation of the kidneys after subcutaneous injection; when internally administered, an excitation of the alimentary canal.

613. Post-mortem appearances. — Post-mortem appearances were fullness in the right side of the heart, intense congestion in the intestinal canal; the stomach, kidneys, and other organs appeared healthy.

XXX. Cedar oil.

614. In general.—The oil of the common juniper (Juniperus Virginiana) has an action upon the system similar to that of savin, except that it appears to have a more decided narcotic influence. Dr. Wait reports four cases of poisoning by this oil, two of which proved fatal. The quantity taken in each case was from half an ounce to an ounce, and in three of them it was swallowed with the view of bringing on abortion. The patients were seized with convulsions, and vomited a fluid having a strong odor of the oil. After the convulsions had subsided, they fell into a comatose condition. The post-mortem appearances in the two fatal cases were not very striking. There were several small, red patches upon the lining membrane of the stomach, and the duodenum showed marked signs of inflammation. The uterus in each case was in a healthy, gravid state. The odor of the oil was distinctly perceived on opening the stomach.[16]

615. Detection.—There are no satisfactory chemical tests for de-

[15] J. Hoppe, Neuenwirkung der Heilmittel, h. 4. 37.

[16] Boston Med. and Surg. Jour. 1849, p. 491.

termining this oil with certainty, and more reliance is to be placed upon the microscopical examination of the contents of the stomach.

XXXI. Savin.

616. In general.—Savin (Sabina, U. S. P.), the tops of Juniperus Sabina, Linné; fluid extract of savin, savin cerate, and oil of savin.

The leaves of this plant have, in the fresh state, a strong, peculiar, and heavy odor,—especially when rubbed,—and a nauseous, resinous, and bitter taste. The dried tops are of a yellowish-green color, and are less odorous than the fresh ones. The oil of savin is a limpid, almost colorless liquid, having the unpleasant odor of the plant, and a bitter, acrid taste. The medicinal dose is from 2 to 6 drops. But the use of the oil or of the dried leaves of this plant in medicine is exceedingly restricted. From the frequency, however, with which it is resorted to for the purpose of procuring abortion, it is necessary to notice its effects. The oil of savin and the fresh leaves, when applied to the skin, exercise a powerful rubefacient and even vesicant action. Swallowed in large doses, it occasions vomiting, purging, and other symptoms of gastrointestinal inflammation. Administered internally in small quantity in the form of the infusion of the leaves or of the essential oil, the tongue perceives a peppery or bitter taste, which is followed by a persistent sensation of freshness on the lips, and by slight twinges in the stomach.[17] The irritating or pungent juice seems to reside in a little gland situated on the back of the leaves in a shallow groove. When administered in large doses, a fluidram (4 c.c.) or more, signs of irritant poisoning are manifested, such as heat at the stomach, epigastric and abdominal pains, vomiting of greenish matters, abundant and often bloody stools, flow of saliva, and in fact the well-known signs of gastrointestinal irritation. The nervous phenomena are those of a general stimulation followed by prostration and collapse. The circulation is at first excited and then becomes feeble, accompanied by a sensation of cold, with a rapid and small thready pulse. The respiration, at first hurried, becomes slow and stertorous, and insensibility then succeeds, sometimes preceded by convulsions. In the first period of excitement or stimulation, the flow of urine is increased and the strong odor of savin is quite distinct in it; oftentimes the urine contains blood, and hemorrhage may occur from nose or lungs; but the congestion is principally noted in

[17] Hamelin. Dict. Enc. des Sciences Médicales. Paris, 1878.

the genito-urinary apparatus, and hemorrhage may take place from the uterus, and, in the pregnant female, the fetus may be expelled, though this does not always happen. It seems pretty evident that abortion does not occur except after dangerous and fatal doses, as suggested by Tardieu and Hamelin; yet the researches of Goltz, Schlœsinger, Hoffmann, and Van Basch, ascribe to savin the property of a direct stimulation upon the uterine organs through the spinal nerve center. Whatever may be the explanation of its action, there is no doubt of its causing a decided local congestion of the pelvic viscera and the genito-urinary organs, from which there is a relaxed condition of the involuntary muscular fibers, differing in this latter respect from rue, which causes strong contraction of the muscles. Pereira says that, according to his observation, it is the most certain and powerful emmenagogue of the whole materia medica. He quotes, from a German author, the case of a woman who swallowed an infusion of savin to occasion abortion. Violent and incessant vomiting was induced, which was followed in a few days by excruciating pain in the abdomen, abortion, dreadful hemorrhage from the uterus, and death.

Two other fatal cases are given by Dr. Christison, in one of which abortion was produced. In two others, related by Dr. Taylor, the women being, respectively, in the seventh and the eighth month of pregnancy, violent and fatal gastrointestinal inflammation was induced and abortion followed. In one of these cases, furnished by Mr. Letheby, the symptoms resembled those of narcotic poisoning; the woman was found lying on her back, perfectly insensible, and breathing stertorously. Although the power of producing abortion cannot be denied to this drug, it is the result of general observation that this effect ensues only when it is taken in such doses as to endanger life by the violent inflammation set up in the stomach and intestines, and that it may even then destroy the life of a pregnant female, without bringing on the premature expulsion of the child. In a case reported by Dr. Hinds, a woman five or six months advanced in pregnancy brought on premature labor by repeated doses of oil of savin. Before and after this event she suffered greatly from purging, vomiting, and intense pain, which terminated in a dangerous attack of peritonitis. She was actively treated, and recovered.[18]

Mitscherlich has made some experiments on animals,[19] which show that the oil of savin is a powerful poison. It is absorbed into the

[18] Times and Gazette, Nov. 1857, p. 524.

[19] Mitscherlich, II. p. 659.

system, as he determined from its peculiar odor, which he detected in the exhalations from the lungs, in the cavities of the body, in the blood, and in the urine. The blood vessels of the intestinal canal, after death from this drug, were injected, and the epithelial coat was in places thrown off; but, according to Mitscherlich, the signs of inflammation in the alimentary canal were not so great as to induce the belief that death from poisoning by the oil of savin was induced by its local action; he inclined to attribute the fatal results to the absorption of the poison itself. He noticed, also, a congestion of the kidneys, and that muscular irritability (contractility) continued for a long time after death.

There is a case, related by Kopp,[20] of an infant who died from the effects of breathing an atmosphere charged with the vapor of oil of savin, which had been left all night in a bottle uncorked near the cradle.

The activity of savin depends upon its volatile oil; the dried drug is less efficient than the fresh.

617. **Symptoms.**—The symptoms produced by savin are those of irritation of the alimentary canal, as also of the kidneys and bladder, together with, in extreme cases, difficulty of breathing, general anesthesia, convulsions, and coma. Death may take place from large doses in twelve to fourteen hours. Its medicinal dose is from 5 to 15 grains (.33 to 1 gm.). The oil of savin quickens the pulse, and induces irritation of the bladder and tenesmus in the rectum. It is eliminated by the skin, breath, and urine. Larger doses cause continued vomiting. Blyth[21] reports a case which came under his own eye: The symptoms of fatal poisoning were preceded by nausea, great pain over the abdomen, and diarrhea, followed by death in twenty-six hours. In this case the savin tops were taken in strong infusion, strength not known, and produced congestion of the gullet and inflammation of the stomach, which contained some greenish matter, probably from the infusion, as the author detected savin tops, and also separated, by distillation, a few drops of a strong, savin-like smelling oil. Death may occur in twelve hours, or may be postponed for two or three days. It is mentioned by Falck, in his toxicology, that commonly the symptoms do not commence for several hours. It has sometimes been used as an abortifacient; but in these cases death has often occurred to the mother as well as to the child, and is prob-

[20] Richter, Ausfürlich, Arzneim, Bd. 408.

[21] Op. cit. p. 559.

ably caused by the irritant action, and congestion of the pelvic organs in general. In many cases death has occurred without expulsion of the fetus.

618. Post-mortem appearances.—After death there are found, in general, undoubted evidences of inflammation of the stomach and intestines. In one of Dr. Christison's cases, the inside of the stomach was red, with patches of florid extravasation, and there was extensive peritoneal inflammation, with fibrinous effusion. The contents of the stomach had a green color. In the case communicated by Dr. Salisbury to Dr. Beck, where the examination was made from twelve to fourteen hours after death, the stomach was found softened and perforated, its contents emptied into the cavity of the abdomen, and there were signs of extensive peritonitis. The perforation was about the size of a fifty-cent piece, and was situated in the region of the greater curvature, near the cardiac orifice. For several inches around the perforation, the stomach was very much corroded, thinned, and softened, so that it was easily torn. The œsophagus and upper part of the small intestine were described as inflamed. Evidence of the presence of savin in the intestinal canal was obtained, and a vial was discovered in the room, still containing ½ dram of the oil of savin and tincture of lavender. In a case occurring to Mr. Lord, of Hampstead, the œsophagus presented a dark, arborescent injection, with slight patches of ecchymosis, and in the stomach there was a large patch of redness about 3 inches in length; the vessels of the mucous membrane were considerably injected, forming infiltrated patches,—especially about the lesser curvature,—but there was no ulceration or erosion. Here, also, a large quantity of a greenish fluid was found, of the appearance and consistency of green pea soup, which was found, on examination under the microscope, to be due to the presence of finely triturated savin powder. The intestines, also, were highly inflamed, the duodenum being of the color of cinnabar, and there was also some peritonitis.

619. Detection.—According to Pereira, powdered savin may, on account of its green color, be mistaken for bile; but when mixed with distilled water, it entirely subsides, and provided no bile be intermixed, the supernatant liquor will be devoid of a green color.

If savin has been given in the form of infusion or decoction, it may be impossible to detect it; but when the oil has been administered, it may be separated by distillation. Furthermore, it has already been stated, savin, in powder, may be recognized by means of

the microscope, the circular pores and the acuminated shape of the leaves being visible. The odor, also, may aid in its recognition.

XXXII. Taxus baccata (yew).

620. In general.—The leaves and berries of the common yew have been known for ages as poisons. Although Orfila gave them to animals in many cases without effect, numerous cases of accidental poisoning by them are known. It is usually classed among the acrid narcotics, although, in most cases of poisoning by it which have been reported, acridity has appeared to be the least essential of its properties. In the case of a lunatic, who died fourteen hours after chewing yew leaves, the symptoms were giddiness, sudden prostration of strength, vomiting, coldness of the surface, spasms, and irregular action of the heart.[22] Similar effects were seen in a child who died four hours after eating the berries.[23] Brandis says a young woman took, as an abortive, the leaves of the yew, and fell into the sleep of death without convulsions. Indeed, one might have supposed her to be really sleeping, for her cheeks preserved the hue of life, and a quiet smile played over her face.[24] In Henke's Journal,[25] an interesting history is given of the poisoning of eleven persons by a decoction of yew leaves. They had partaken of it as a prophylactic against hydrophobia, some of their dogs having been bitten by one supposed to be rabid. In half an hour all of them were seized with giddiness, confusion of sight, pain in the head, nausea, and vomiting, and then they fell asleep. Two of them, however, died within about an hour, without either pain or convulsions, but with a smile upon their countenances. The rest recovered without further symptoms. The post-mortem appearances, in these and the preceding cases, threw no light upon the manner in which the poison affected the system, except from the negative evidence of the absence of any well-marked signs of inflammation. In Mr. Hurt's case, however, it is stated that, besides patches of redness upon the mucous membrane of the stomach, it was also much softened.

XXXIII. Sulphonal.

621. In general.—Sulphonal is the proprietary name of diethyl

[22] Dr. Mullan, Dub. Hosp. Gaz. 1845, p. 494.
[23] Hurt, Lancet, Dec. 10, 1836.
[24] Blumenbach's Med. Bibliothek, Bd. III. p. 684.
[25] Erg. Heft, 43, p. 127.

sulphon-dimethyl-methane. It is a colorless, odorless, almost tasteless, crystalline body (m. p. 125°C.). It is fairly soluble in boiling water, very slightly in cold water, freely soluble in hot alcohol.

Sulphonal has been used extensively during the last ten years as a hypnotic drug, and is a derivative of methane. In large doses it is a poison.

622. Symptoms.—The symptoms are sleep deepening into coma, and accompanied with clammy skin; slowing of respiration and circulation; sometimes, increase of bodily temperature[26] reaching to 103°F.; the urine sometimes entirely suppressed, but more generally has a reddish-brown or reddish-black color, owing to the presence of hematoporphyrin. Symptoms of ataxia with muscular tremblings appear later. Vomiting has occasionally occurred before the tendency to sleep. In very severe cases there is stertorous breathing, the pulse scarcely perceptible, and with marked cyanosis. Death usually results from asphyxia, but artificial respiration does not appear to prolong life. Occasionally, eruptions of the skin may follow a single large dose. Sleep and coma have persisted for six days, and recovery taken place.

623. Chronic poisoning.—The continued use of sulphonal to procure sleep, when persisted in for a long period of time, produces a form of chronic poisoning; the symptoms of which are headache; vomiting; albuminuria; suppression and the peculiar discoloration of the urine above noted. In addition to these symptoms there may be various mental disturbances, as well as ataxic symptoms, with the various forms of skin eruption described by the observers.[27]

624. Lethal dose.—The effect on different people who have taken sulphonal shows that there is great latitude in regard to the size of the dose which may produce poisonous symptoms; for instance, Kobert[28] described a case of slow sulphonal poisoning, the dose varying from 7 to 22 grains (.45 to 1.38 gm.) for a period of four to five weeks; during this course of treatment the urine gradually changed to the Burgundy-red above described, and, finally, reddish-black color; retention of the urine, and then death occurred. Bresslauer[29] published in Vienna a series of cases of 27 insane patients to whom doses of sulphonal were administered for a considerable time, of whom 7 showed serious symptoms, and 5 of these died. The drug had been borne well at first, and the serious symptoms appeared sud-

[26] Knagg's Brit. Med. Jour. 1890.
[27] Politz, Wien klin. Wochenschr. 1898, Vol. II. p. 566.
[28] Centralblatt f. klin. Med. 1892.
[29] London Lancet, 1891.

denly; these were constipation, dark-colored urine, feeble pulse, and great prostration; apparent cause of death was heart failure with edema of the lungs. Two cases recorded by Grant[30] are described as having alarming symptoms of muscular weakness and prostration, produced after single doses of 20 grains (1.3 gm.); 30 grains (1.94 gm.) caused death in a feeble woman in forty hours (Pollitz). Death has frequently followed doses ranging from 75 grains (4.85 gm.) to 1 ounce (31 gm.); on the other hand, a boy of fifteen years of age recovered after a dose of 3 ounces (93 gm.), being ninety hours in continuous sleep. Death may occur either within a few hours or after weeks or months. In the latter case, after continued administration of small doses, and even after they have been stopped, death has occurred quite a long time later.

625. Treatment.—In acute poisoning, washing out of the stomach; stimulating the urinary secretion by diuretics, the bowels by purgatives; strychnin to increase the action of the involuntary muscles in chronic cases. Caution should be taken to administer sulphonal in hot water to secure its more rapid absorption, and its administration should be interrupted every few days. Kobert recommends the inhalation of ammonia for the relief of poisonous symptoms of sulphonal, as also the injection of ether, camphor, or digitalis, subcutaneously.

626. Action.—Kobert gives the action of sulphonal and trional as strong narcotics. The peculiar red color found in the urine after taking sulphonal is due to the destruction of the coloring matter of the blood, and its elimination by the kidneys. It would seem from what we can learn from the reports in medical literature that, after the action upon the blood in disorganizing its coloring matter, the passage through the urinary organs produces an irritation which may obstruct the elimination; and this explains the danger of continuing the drug, on account of its interference with the functions of the kidneys showing the so-called "cumulative" action.

627. Post-mortem appearances.—The action of the allied substances of the methyl groups to which sulphonal belongs should comprise the other two relations of that group, called "trional" and "tetronal," the symptoms of which are similar to those caused by sulphonal.

628. Detection and tests.—Upon heating sulphonal with KCN the odor of mercaptan develops, and KCNS is also formed. The fused mass, on solution, yields a red color, when treated with ferric chlorid.

[30] Boston Med. and Surg. Jour. 1892.

Heated with powdered charcoal, sulphonal gives off mercaptan, sulphur dioxid, acetic acid, and formic acid. The mercaptan may be recognized by its odor, the acids by turning blue litmus paper red, and the SO_2 by its bleaching properties.

Material to be tested for sulphonal is first extracted with alcohol, the alcohol evaporated, the residue extracted with boiling water, evaporating the water, and extracting this residue with ether. The ether, on evaporation, leaves the sulphonal as a residue, which may be crystallized from boiling water, and a determination of its melting point made.

XXXIV. Trional and tetronal.

629. In general.—If one of the methyl groups of sulphonal is replaced by an ethyl group, trional is formed; if both methyl groups are so replaced, tetronal results. They are very similar to sulphonal in their properties and action.

XXXV. Tansy.

630. Properties.—Tansy contains a volatile oil. It is used occasionally as an anthelmintic. Its action is similar to oil of turpentine, savin, etc.

631. Deaths from use of.—In the Massachusetts medical examiners' returns there are 2 cases of death by suicide, where tansy was taken for the purpose of producing abortion. Tansy leaves are poisonous.

632. Symptoms of poisoning.—The symptoms of poisoning are similar to those caused by savin, such as collapse, cyanosis, and narcotism, followed by convulsions. A patient died twenty-six hours after taking an infusion of about a half ounce of the dried herb.[31]

Oil of tansy has often been taken for the purpose of inducing abortion, but does not seem to possess this property, which is popularly attributed to it. A fatal case of poisoning with half an ounce of this oil is recorded in the American Journal of Medical Science for May, 1835. Frequent and violent clonic spasms were experienced, with much disturbance of respiration. No signs of inflammation in the stomach and bowels were found upon section. Death occurred in two hours after taking the poison. Other more recent cases of fatal poisoning are recorded.[32]

[31] Phila. Med. Times, 1881, Vol. XX. p. 346.

[32] Thérap. Gaz. 1885, Vol. IX. p. 342; Phila. Times, 1881, Vol. IX. p. 346.

The symptoms of poisoning by the oil of tansy are a sense of heat and discomfort in the stomach and bowels, attended with giddiness, muscular weakness, prostration, disturbances of the circulation and respiration similar to those observed after the other previously described essential oils, death taking place, after coma, from asphyxia, though sometimes preceded by convulsions.

There have been quite a number of deaths caused by this oil, taken inadvertently, or for the purpose of promoting the menstrual discharge.[33]

A young lady took a teaspoonful of the oil by mistake for the essence of tansy, for the purpose of promoting the catamenial discharge. She complained of dizziness, became insensible in ten minutes, and was seized with convulsions; her respiration was laborious and her pulse irregular; she died in one and a quarter hours after taking the oil. Another young lady of the family took of the medicine at the same time, but vomited very soon, and suffered no inconvenience.[34] In a case which came under the notice of Dr. Dalton, of Lowell, recovery took place, in consequence of spontaneous vomiting having occurred. Nevertheless the girl remained insensible and convulsed for some time after it. The most interesting and detailed case is that related by Dr. Dalton, Jr., of Boston. The quantity taken was a little less than an ounce and a half, and death took place in three and a half hours. The girl, when first seen, had fallen out of bed, in convulsions, and was entirely unconscious. The cheeks were highly flushed, the eyes open and brilliant, the pupils widely dilated and insensible; the skin was warm; pulse full, rapid, and strong; respiration hurried and stertorous, and obstructed by an abundance of frothy mucus, which filled the air passages and was blown from between the lips in respiration; the breath had a strong odor of tansy; convulsions occurred every five or ten minutes, in which the respiration was suspended, the arms raised and rigidly extended, and the fingers contracted. In the intervals between the convulsions there was no return of consciousness, and the jaws remained clinched, so that it was impossible to administer any medicine by the mouth.

Half an ounce of oil of tansy, taken by a pregnant female to pro-

[33] Smith, Detroit Lancet, 1881–82, 147; Wetzell, Am. Med. Biweekly, Louisville, 1879, 58; Stephens, Nashville Med. & Surg. Jour. 1879, 320; Jewett, Boston Med. & Surg. Jour. 1880, 237; Stuart, Peoria Med. Monthly, 1881–82, 294; Pittsburgh Med. Jour. 1880–81, 22; Tidd, Detroit Lancet, 1881–82.

[34] Am. Jour. Med. Sci. July, 1852, p. 279.

cure abortion, produced a partial loss of consciousness, and convulsions. It failed, however, of its purpose.[35]

633. Post-mortem appearances.—The autopsy of the case of Dr. Dalton, above recited, is given as follows: There was no particular characteristic sign of the poison producing pathological conditions.

The examination was made ten hours after death. The countenance was natural, the cadaveric rigidity was very strong, and there was only a slight discoloration of the dependent parts. The brain was not congested in any part, nor was there any effusion. Neither was there any appearance of congestion in the lungs. The interior of the heart exhaled a distinct odor of tansy, as did also the cut surface of the pectoral muscles. There was a strong odor of tansy in the peritoneal cavity. "The stomach contained about 12 ounces of a semifluid, yellowish-gray substance, consisting of partially digested food—potato, cranberries, onions, etc.—mixed with an abundance of small, yellowish-brown, glistening oil globules, exhaling a powerful odor of tansy; mucous membrane generally pale, not vascular in any part, but, throughout nearly the whole of the great pouch, brownish and much thinned and softened, so that for a considerable space it was nearly or quite destroyed. There was an old, whitish, slightly puckered cicatrix of the mucous membrane on the posterior wall of the stomach, near the smaller curvature, but no other morbid appearance."[36] A four months' fetus was found in the womb, not in the least disturbed. A two-ounce vial, still containing 5 drams of the oil of tansy, was found in the pocket of the girl's dress; and a mug, from which it had apparently been drunk, mixed with water, was also found, smelling very strongly of the medicine.

In the two cases of suicide returned by the Massachusetts medical examiners in 1892 and 1896, the autopsies showed no particular, characteristic pathological conditions of the viscera, except slight inflammation of the stomach in one case, and in the other, pelvic peritonitis (nonpurulent hemorrhage). Only the uterine organs and peritoneum showed signs of disease.

634. Detection.—No other means have as yet been proposed, or are perhaps necessary, for the detection of this oil in cases of poisoning by it, than its powerful and peculiar aromatic smell.

[35] Boston Med. and Surg. Jour. Dec. 1857, p. 383.

[36] Those appearances would hardly be called morbid, but probably were due to a post-mortem digestion of the stomach by its own secretion. Ed. 4th ed.

XXXVI. Turpentine.

635. Properties.—Oil of turpentine is a volatile oil distilled from turpentine, and an oleoresin obtained from Pinus palustris, and other species of Pinus. It is a mobile, colorless liquid, boiling at 160°. It is almost insoluble in water, but dissolves in alcohol, ether, and glacial acetic acid. It has a hot, burning taste; and applied to the skin, causes irritation and redness. It is extensively used in the arts as a solvent.

636. Poisoning from use of.—Poisoning has resulted from its use in medicine, *e. g.*, anthelmintic. It has also been the cause of accidental or suicidal death, but has rarely been used for homicidal purposes.

The Massachusetts medical examiners' returns show one death by homicide in 1878; two cases of suicide recorded in 1892 and 1897. One of the cases of suicide was of an insane person; amount swallowed not stated. The case of homicide was of a woman, whose husband confessed to having given her turpentine to produce abortion.

637. Acute symptoms of poisoning.—After a fatal dose of oil of turpentine, the following symptoms are generally produced: Burning pain in mouth and throat, followed by vomiting and diarrhea; clammy surface of the body, and narcotism, with difficult micturition, sometimes with strangury and bloody urine; the urine has a violet-like odor; muscular spasms and coma, followed by death.

638. Subacute symptoms of poisoning.—These may arise from inhaling the vapor of turpentine, as, for example, where persons are sleeping in a room newly painted with preparations containing oil of turpentine, or in factories where turpentine is used. The symptoms in these cases are dizziness, mental depression, painful micturition, bloody urine, the appearance of albuminuria, and the violet-like odor of the urine passed.

639. Lethal dose.—To a child under six months, a tablespoonful of the oil of turpentine; to an adult 6 ounces (186 c.c.).

640. Treatment.—Evacuation of the stomach, cardiac stimulants, and mucilaginous drinks to prevent the irritation of the urinary system.

641. Detection and tests.—There are no conclusive, chemical tests that can be used to very satisfactory advantage; but a few of its reactions may be of assistance in determining its presence: A paper dipped in turpentine and introduced into a jar of chlorin gas in-

flames spontaneously, forming substitution products. Iodin and bromin have a similar action upon it. It is also very inflammable.

XXXVII. Amyl nitrite; nitroglycerin; spiritus glonoini.

642. Properties.—Amyl nitrite, $C_5H_{11}NO_2$, is a colorless, volatile liquid with a peculiar odor (b. p. 96°C.). It is insoluble in water, soluble in ether. This is a limpid and, generally, slightly yellow liquid, and has a peculiar and characteristic odor. Heating amyl nitrite with alcoholic potash will produce amyl alcohol and nitrite of potash.

643. Symptoms and action.—The nitrite as well as the acetate and the iodid of amyl have the same action upon animals. The heart acts violently, and there is at first a dilatation of the capillary blood vessels, which is soon followed by a diminished action of the heart and contraction of the capillaries. Its action on frogs will suspend animation, the animal appearing to be dead for many days and yet recovering. On warm-blooded animals a condition of catalepsy is produced by amyl nitrite. It is not an anesthetic, and it does not produce unconsciousness, except when apparent condition of death is first produced. When the latter occurs there is rarely recovery, the animal passing into actual death. After large doses, symptoms of unconsciousness appear, with vomiting and with shallow respiration; cyanosis may precede collapse, which is due to the formation of methemoglobin in the blood, causing the latter to assume a chocolate color. Headache, and confusion of mental action, are often present, and a peculiar flush of the countenance,—especially when its vapor is inhaled. It is not a particularly dangerous poison, unless its administration is continued too long.

644. Nitroglycerin in general.—Nitroglycerin (spiritus glonoini), trinitrate of glycerin, $C_3H_5(NO_3)_3$, is a pale yellow, oily liquid, nearly insoluble in water, but soluble in absolute alcohol, ether, and chloroform.

Nitroglycerin is a substitution compound formed by the action of nitric acid on glycerin. According to Dr. Taylor,[37] this heavy liquid has been used in Sweden in mining, under the name of "blasting oil," and in some instances, by mistake, has been taken by the miners with serious consequences. In one case a miner swallowed "two mouthfuls," which was followed by a painful feeling in his throat, after which he drank a quantity of milk; when seen by a physician in an

[37] Treatise on Poisons, Phila. p. 642.

hour and a quarter after swallowing the liquid, he was suffering with faintness, difficult breathing, and oppression at the chest. In five hours, vomiting and purging set in, and shortly before death the lips became livid, and the man lay quietly as if asleep, breathing feebly, and occasionally with a deep sigh. The post-mortem examination revealed great congestion of the brain, as well as of the lower lobes of the lungs. The lining membrane in the air passages had a reddish-brown tint. The greater curvature of the stomach presented a similar appearance, with some ecchymosis.[38] A case, attended by serious symptoms caused by homeopathic pellets of glonoin (supposed to be the first trituration), was observed by the writer. As nearly as could be ascertained, about thirty to fifty of these pellets were swallowed by a boy three years of age, who was found lying on the floor, conscious and calm. He was seen by the writer within twenty minutes after the time he was supposed to have swallowed the pellets; he was lying upon a sofa with pallid but peaceful countenance, feeble, rapid pulse, and slow movements in breathing, which were not full, but shallow. He could not cause any contraction of the muscles below the waist, and could not stand. He complained of a slight headache, but there was no other apparent symptom. No remedy was given, but in six or seven hours he was up on his legs, and had no further serious trouble.

645. **Action of nitroglycerin.**—Nitroglycerin produces the same action as amyl nitrite, because it is decomposed into a mixture of potassium nitrate and nitrite, the peculiar effect of the respiration and circulation being due to the nitrite which is formed. After absorption into the blood, amyl nitrite undergoes partial oxidation, and will appear in the urine in the form of nitrates and nitrites; but the quantity of these excreted is never equal to the amount of nitrite absorbed.

Some of the amyl nitrite is exhaled with the breath. When swallowed, nitrite of amyl gives off nitrous acid, from the action of the gastric juice, and generally gives rise to glycosuria and slight diuresis. It has been assumed that the formation of methemoglobin may cause partial nonoxidation of the tissues, thus causing the formation of an excess of lactic acid and glycosuria.

646. **Lethal dose.**—A single dose of 10 drops inhaled without air would probably be fatal, though the recorded deaths are generally from larger doses,—especially when swallowed. In factories where nitroglycerin, etc., are made, workmen acquire a tolerance; and pa-

[38] Dr. Taylor has taken the above account from Husemann's Jahresbericht, 1872, p. 533.

tients, habituated to this poison used in small doses during a long period of time, as, for instance, twelve months, have taken 6 grains (0.4 gm.), without fatal consequences.

647. Treatment.—Washing out the stomach, and administration of cathartics to empty the bowels, if the poison has been swallowed; if taken by inhalation, artificial respiration and inhalation of oxygen should be tried; probably the transfusion of a saline solution, after a vein has been opened to let out the dark-colored blood, under venous stasis, might stimulate the respiratory and cardiac actions.

648. Post-mortem appearances.—When quickly administered in large doses, the lungs and other internal organs appear whitened and free from blood, the right side of the heart gorged, the left empty, and the brain blanched. When slowly administered in small doses, the brain is found suffused with blood, and there is blood both in the left and right sides of the heart.

The medicinal dose of this solution is stated as ½ to 2 or 3 drops; a single drop has caused unconsciousness, abolition of pulse, and insensibility, followed by rapid recovery.[39] The effect of inhalation of the volatile nitrites is intense suffusion of face, fullness of head, and unconsciousness; at first, the pulse is very rapid and tumultuous, though feeble; but the blood pressure is weak, and the bodily temperature remarkably lowered. These symptoms increase for a few moments after the inhalation has ceased. Though the effects of nitrite of amyl and other volatile nitrites are very rapid and, apparently, severe, instances of death following their use are rare, and there is no recorded case of their criminal use. Death from this class of drugs would be caused by asphyxia, and the post-mortem appearances in the blood, as shown by the spectroscope, are due to this cause, the chocolate-colored blood from poisoned animals showing, in the spectrum, the absorption bands of methemoglobin.[40]

649. Detection.—It is very volatile, and care must be taken in distilling it from the material under investigation lest some of it escape. On heating with potassium hydrate solution, it is decomposed into amyl alcohol and potassium nitrite. The presence of the latter can readily be confirmed by many tests.

[39] Brit. Med. Jour. 1880, I. 408.

[40] Hoppe-Seyler, Zeitsch. für Physiol. Chem. Bd. II. u. III., quoted by Dr. Wood in his Mat. Med. and Toxicology, Phila. 4th ed. p. 359. For detailed and elaborate experimental studies with amyl nitrite, reference is made to the following works: T. Laudor Brunton, Jour. of Phys. and Anat. Vol. V.; H. C. Wood, Am. Jour. of Med. Sci. July, 1871; Amez. Dros, Archives de Phys. Normale et Pathologique, Sept. 1873; Pick Central, F. D. Med. Wissen. 1873.

CHAPTER V.

ALKALOIDS.

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I. Aconitin (monkshood; wolfsbane).

650. Occurrence.—The leaves and root of Aconitum Napellus contain one of the most extraordinary and fatal poisons known. The former have proved fatal when eaten by mistake for salad;[1] and the latter, from its resemblance to horse-radish, has given rise to many unfortunate accidents. The root is tapering, about the thickness of the finger at its upper part. Its color, externally, is brown; internally, it is white and fleshy. Its taste is bitter; but, after a few minutes, a marked numbness and tingling are perceived on the lips, tongue, and fauces. The leaves, when chewed, have the same taste, and produce the same feeling of numbness.

Aconitin is the chief of the poisonous alkaloids of Aconitum Napellus, known as the common monkshood. Aconitum contains three or four alkaloids,—aconin, napellin [nepalin] lycaconitin, and myoctonin; the last two being amorphous, very poisonous alkaloids, obtained by Dragendorff from Aconitum lycoctonum. Inasmuch as the chemistry of these various alkaloids is at present so undecided,

[1] Pereira. For several cases, *vide* Headland, Lancet, March, 1856, p. 341.

and as there is also some question as to whether other alkaloids that have been found by other investigators may not be derivatives of aconitin, and as the symptoms produced are very similar, it is hard to differentiate between them all, and a discussion of aconitin may be taken to be typical in most respects for the others.

651. Properties.—Aconitin is a crystalline alkaloid, the exact formula being probably either $C_{33}H_{45}NO_{12}$ or $C_{34}H_{47}NO_{11}$. It fuses at 188.5°, soluble in about four parts of water, easily soluble in chloroform and benzol, somewhat so in alcohol and ether. It is easily decomposed by acids, alkalies, and, even on boiling, in the presence of other substances, which makes its detection, in a systematic analysis, difficult. It gives no distinctive chemical color tests, and its detection in organic mixtures is chiefly dependent upon its physiological action.

652. Preparations.—All of the preparations of the Aconitum Napellus which are officinal (U. S. P.) are now made from the root, and not from the leaf or plant. The alkaloids are not recognized as officinal (U. S. P.).

653. Statistics of poisoning.—Aconite has been used since 1840 as a poison, with intent to murder. Mr. Blyth's tables of mortality record 40 accidental, and 19 suicidal deaths, in England and Wales, from various forms of aconite; he also "collected, from European medical literature, 87 cases of poisoning by aconite in some form or other, of which two were cases of murder, 7 of suicide, and 77 more or less accidental. Six of these cases were from the use of the alkaloid aconitin, and 10 were from swallowing the root ; in 2 cases, children ate the flowers; in one case, the leaves of the plant were cooked and eaten by mistake ; in 7, the tincture was taken for brandy, sherry, or liqueur; the remaining cases of poisoning were caused by the tincture, liniment, or extract." In 1841 there was reported a case in Ireland, where murder was attributed to aconite root. In this case, a woman (McConkey) was tried for the murder of her husband, to whom she administered, mixed with pepper, some powdered aconite root, which was strewed over some greens which she cooked and gave to him. The husband died in about 3 hours. In 1842, in the East Indies, the natives, while retreating from pursuit of the British army, poisoned a tank of water with the bruised root of Aconitum ferox; this was fortunately discovered before the water was used; this poison was well known to the natives of the hill districts of India, who used the aconite root to poison arrows used in hunting wild beasts.

In the Massachusetts medical examiners' returns there are 14 cases recorded, where some preparation of aconite, sometimes mixed with opium, has been taken to produce suicide by this poison; in one case death occurred eleven hours after ingestion of aconite.

One medical examiner reports that a party of seven tramps drank some alcohol containing tincture of aconite; two died; the five that recovered had drunk sparingly of the liquid. Among the other cases of accidental poisoning was one which was caused by the druggist taking aconite tincture from the shelf, when he intended to take Jamaica ginger.

654. Symptoms of poisoning.—When the preparations of aconite were made from the leaf or the root, dangerous symptoms were more apt to follow excessive doses, because of the unfamiliarity with the strength of the active principle contained in the preparation.

There being several formulæ in different pharmacopœias for these preparations they vary in strength, owing to the variable quantity of aconitin contained in the prescribed dose. Thus, two persons, who took 25 minims of the tincture, died;[2] while another, who swallowed 1½ ounces of the tincture, prepared according to the French Code, survived. The active principle, called "aconitin," which is very variable in its poisonous strength, prepared by Mr. Morson, of London, is so powerful that, according to Pereira, 1/50 of a grain has endangered life; but, on the other hand, a case is reported by Dr. Golding Bird, in which, although 2½ grains of this alkaloid (not Morson's) were taken, the patient recovered, after having very dangerous symptoms. Pereira states, also, that there is a spurious aconitin, which is inert or nearly so, sold in the shops, since he took one grain of it without perceiving the least effect upon the tongue, or otherwise.[3] The effect of a slight increase in the medicinal dose is well seen in the following case, communicated to Pereira by Dr. Redfern: The patient who was suffering from acute articular rheumatism, took 5 drops of the tincture three times a day for two days, without marked relief. On the third day the dose was increased to 6 drops, at the same intervals. Two doses of this amount were taken; and, an hour after the second, he was found in a state of extreme restlessness, complaining of great pain in various parts of his body. To use his own expression, he felt as though his skin were too

[1] The tincture made from the root is about five times stronger than that made from the fresh leaves.

[2] The preparations sold under the name of this alkaloid are altogether unreliable; a number of experiments were conducted by a former editor with a sample which appeared to be not much stronger than the powdered root itself.

tight for his body. He described his sensations as intolerable. At this time there was much frothing at the mouth, with violent retching at intervals. The surface of the body was cold, and bathed in profuse perspiration, which ran down his face in streams. The pulse, though at first 150 a minute, fell to between 50 and 60 in a few minutes, and was so small and compressible as scarcely to be felt at the wrist. He recovered, under the use of brandy and water and external warmth.

The tincture of the leaves is weak and uncertain, and so are, often, the tinctures which are made from the root; they vary in the officinal pharmacopœias. The U. S. P. prescribes 40 parts, by weight, of aconite in preparing 100 parts of tincture; the British Pharmacopœia, 16 parts; the German, 10 parts; Fleming's tincture, 79 parts; and the British liniment of aconite, 108 parts. The officinal tincture varies very much, and this accounts for the difference in the symptoms noted of cases reported where a tincture was used. However the poison is received, whether by chewing the bruised root or the leaves of the plant, or by the tincture in excessive medicinal doses, the peculiar, sweetish taste in the mouth, experienced even from slight doses, is followed immediately by a peculiar tingling of the lips and tongue; and, if the dose is a dangerous one, numbness of the face with severe vomiting follows. This numbness and tingling sensation soon afterwards extends to every part of the body, and the face wears an anxious look, with suffusion of the eyes, paleness of the lips, swollen eyelids, pupils moderately dilated and insensible to the stimulus of light; respiration becomes rapid and shallow, and the pulse weak and feeble; intelligence remains, though the patient may be unable to walk, on account of weakness of the muscles. To all the above symptoms there is a great restlessness superadded. When the symptoms become poisonous, the sense of numbness becomes one of pain; the pulse grows weaker and irregular, and, finally, imperceptible; the face looks pinched and white, the pupils of the eyes are dilated, the surface is cold, and the respiration labored; convulsions may also occur, though the mind is usually clear. These symptoms of poison may be developed with great rapidity, and death occurs within three hours. Associated with the convulsions may be the grinding of the teeth, and difficulty in swallowing and speaking. Usually delirium, as well as stupor, is absent; the respiration becomes slower and slower, and the patient dies from paresis of the respiratory reflex nervous center.

A case of very severe poisoning by taking a mixture of aconite

and chloroform is related in the Appendix, which varies somewhat from the general symptoms above indicated. Other cases are referred to in the footnote.[4]

The first and most usual symptoms are a burning and numbness of the lips, mouth, throat, and stomach, followed by tingling in various parts of the body, loss of sensation, vertigo, dimness of vision, tremors, great prostration, sense of fullness in the throat, speechlessness, hurried respiration, and death in a state of collapse. Vomiting and purging are also usual symptoms, but are not observed in every case. General convulsions are unusual, as we find that, in 53 cases collected by Dr. Tucker, of New York,[5] these are mentioned as having occurred only in 7. In four out of 12 more recent cases there were convulsions. The mind remains perfectly clear, there being, in general, neither stupor nor delirium. The latter symptom was seen in 3 cases of the number collected by Dr. Tucker. When applied to the eye, the preparations of aconite are said to have the effect of contracting the pupil. In 17 out of 20 cases, however, in which the poison was swallowed, it is stated by Dr. Tucker that the opposite effect was observed. Sometimes the sight is temporarily impaired. The symptoms of poisoning by aconite usually arise within a few minutes after it has been taken; and, when death takes place, it is, in the majority of cases, within three hours.

655. Lethal dose.—The quantity of aconite, or any of its preparations, capable of producing death, is, for the reasons before given, unknown. The case related by Dr. Easton,[6] in which 25 minims of the tincture were taken, shows, probably, the smallest dose which has proved fatal. Another case, in which 25 drops proved fatal, occurred in January, 1853. A gentleman, feeling himself unwell, stepped into a drug store, and was given by a medical student, a friend of his, who was attending, this amount of the tincture, under the supposition that it was the proper dose. He expired about four hours after taking it, with the symptoms of poisoning by aconite. Dr. Male, of Birmingham, it is stated, died from the effects of not more than 80 drops, taken in ten doses, during a period of four

[4] Stewart, case of poisoning with aconite and fusel oil, Edin. Med. Jour. 1879, 904; O'Brien, case of poisoning by aconite, Med. Rec. N. Y. 1879, 128; Carleton, case of aconite poisoning, Boston Med. and Surg. Jour. 1879, 544; Elliot, case of aconite poisoning, Lancet, London, 1878, II. 917; Pike, *Ibid.* 1880, I. 12; Hardman, *Ibid.* 1882, I. 37; Samson, a case of poisoning by aconite, Chicago Med. Rev. 1882, 91; Van Peski, Empoisonnement par le nitrate d'aconitine. Gaz. Hebd. de Sci. Méd. Montpellier. 1880, unique case of poisoning by aconite, Brit. Med. Jour. 1881, 64.

[5] For these valuable tables, *vide* New York Jour. of Med. for March, 1854.

[6] Glasgow Med. Jour. for July, 1853.

days, the largest quantity taken at one time being 10 drops. An excise officer, in England, died in consequence of tasting Fleming's tincture of aconite (strength of 79 per cent). He said he thought he had swallowed about a teaspoonful. He did not complain at the time, but, in the course of a few hours, was cold and pulseless. The remedies applied were ineffectual, and he soon expired.[7]

The fatal dose of pure aconitin, which is one of the most powerful known poisons, is less than 1/16 of a grain (0.004 gm.). It would not be safe to administer a dose over 1/32 of a grain (0.002 gm.). The difficulty in stating the exact minimum dose that would be expected to produce dangerous or fatal symptoms is owing to the fact that the preparations of aconitin are not of uniform strength.

656. The aconite alkaloids.—Until recently, the result of preparing an alkaloid from aconite that would represent all of its poisonous properties has been rather unsuccessful; later, however, the researches of Dr. A. Wright and Dr. Luff, and especially those of Prof. Dunstan,[8] show that there are contained in the root of the aconite plant four alkaloids, only one of which has yet been crystallized. Three of the alkaloids are aconitin, aconin, and benzoyl-aconin; the fourth, called "homo-napellin," has not yet been carefully worked out. The separation of these various constituents is mainly determined by their different melting points.

Aconitin has been demonstrated by Prof. Dunstan as acetyl-benzoyl-aconitin, so that 100 parts of aconitin, theoretically, should yield 9.37 per cent of acetic acid, 18.85 per cent of benzoic acid, and 77.52 per cent of aconin. Pure aconitin has a test tube melting point at 188.6° C., and gives no characteristic sublimate, up to 190°. It is sparely soluble in water at 22° C. (1 part in 4,431 of water); it is soluble in 37 parts of absolute alcohol, 64 parts of anhydrous ether, and 5.5 parts of chloroform and benzene. The most satisfactory test for the presence of aconitin would be by application of the minutest trace of an aconite-holding liquid to the tongue or lips; this will cause a peculiar numbing and tingling sensation, which, when once felt, can always be remembered.

Prof. Dunstan[9] has recently examined fourteen samples of commercial aconitin, two samples of which approached, in melting point, a crystalline appearance of pure aconitin. Since that time it has not been difficult to obtain fairly pure crystalline aconitin, and it

[7] Am. Jour. of Med. Sci. April, 1852, p. 553.

[8] Consult Jour. Chem. Soc. 1892–94.

[9] Jour. Chem. Soc. Trans. 1893, 491.

can be assayed accurately by determining the proportion of acetic and benzoic acids. The physiological action of commercial aconitin, however, should always be the same, the strength of its action being dependent upon the amount of true aconitin present in the sample used, the other basis being practically without toxic action.

From experiments on animals, Prof. Plugge[10] has concluded that Petit's crystalline nitrate of aconitin is at least eight times stronger than that of Merck, and seventy times more toxic than that of Friedländer, while Merck's aconitin is from twenty to thirty times stronger than Friedländer's. He would place seven commercial samples, which he has examined, in the following order of toxicity: (1) Petit's crystalline aconitin nitrate; (2) Morson's aconitin nitrate; (3) Hottot's aconitin nitrate; (4) Hopkins and Williams' pseudo-aconitin; (5) Merck's aconitin nitrate; (6) Schuchart's aconitin sulphate; and (7) Friedländer's aconitin nitrate.

Man is more sensitive to the action of aconitin than the dogs or cats submitted to experiments, and it will be seen from the following cases of poisoning, which have happened in Germany, that 1.6 milligrams of Petit's aconitin nitrate, taken by the mouth, gave rise to symptoms so violent that it was evidently a dangerous dose, while 4 milligrams were rapidly fatal; but, if man could take the same amount per kilo weight as dogs or cats, it would require a little over 6 milligrams to be certainly fatal. Mr. Blyth, therefore, concludes, "from the evidence obtainable, that 3/100 of a grain (.0002 gm.) is about the least fatal dose for an adult of standard weight. This dose is equal to about .028 milligrams per kilo, and, of course, refers to either Morson's aconitin or the French aconitin, the alkaloid being taken by the mouth. If given by subcutaneous injection, probably 1.5 milligrams would kill; for the whole of the poison is then thrown into the circulation at one time, and there is no chance for its elimination by vomiting."

The tincture of aconite of the British Pharmacopœia, although variable in strength, may, for our purposes, be regarded as averaging 1 per cent of alkaloid; Fleming's tincture should be considered as ⅓ stronger, or equivalent to 1.3 parts of alkaloid to 100 parts of the tincture. The medicinal dose of the British Pharmacopœia tincture, being laid down as 5 to 15 minims, would, therefore, be equal to from 5/1000 to 15/1000 of a grain of aconitin. The usual least fatal dose, laid down in medical toxicological books, is, however, greater than this; *viz.*, 1 dram (3.88 c.c.) of the tincture of aconite

[10] Archiv. de Pharm. Jan. 7, 1882.

(Br. P.). In a case quoted by Taylor (Vol. I. p. 426), the smallest quantity of the tincture (Br. P.) of aconite, which is reported as having destroyed life, was 80 drops, taken in ten doses during a period of four days, the largest dose being ten drops; the total quantity taken would, perhaps, be equal to 8/100 of a grain of aconitin. In regard to the root of aconite, 60 grains (3.9 gm.) have been known to produce death.

657. **Treatment.**—Evacuation of the stomach by the stomach pump; tannic acid or finely powdered charcoal should first be administered, in order to produce an insoluble form of the alkaloid; heart stimulants should then be given, such as carbonate of ammonia, spirits, and, perhaps, if care is used, the subcutaneous injection of digitalis and strychnin. Above all means, the patient's body surface should be kept warm. In the case mentioned in our Appendix, great benefit resulted from electrical irritation after the cessation of pulsation in the arteries, even in the axilla, and by artificial respiration, also by keeping the head low. The electrical stimulation, in the case mentioned, was continued at times during the succeeding eleven hours; "when discontinued for more than ten minutes, the pulse rate would flag at once, and coma come on."

658. **Action.**—Aconitin possesses, in a large measure, many of the poisonous characteristics of aconite, and is, according to Dr. Ringer, a protoplasmic poison, suspending the functions of all nitrogenous tissue. According to this observer, first the functions of the central nervous system are inhibited, next those of the nerves, and last, the contractility of the muscles. Without a doubt, aconitin acts powerfully upon the heart, which it finally paralyzes; this effect, as observed, would indicate, at first, a slowing of the pulse, due to excitation of the central portion of the vagus; finally, the heart's pulsation becomes slow, irregular, and weak, and the arterial blood pressure sinks. There is lack of agreement as to whether dyspnea, convulsions, and the failure of respiratory movements, are the consequence of the weakened action of the heart, or whether the motor paralysis has a central origin, by means of which the functions of these organs are suspended.

The present weight of evidence seems to indicate that the action is like that of curare,—that the muscles themselves preserve their irritability; and that the paralysis of respiratory motion and of the pulsation of the heart is due, first, to the irritation, and then to the consequent exhaustion, of the vagus nerve or reflex nerve center.

659. **Post-mortem appearances.**—The most common post-mortem

appearances, among the warm-blooded animals, have been hyperemia of the cerebral membranes and of the brain, and fullness of the large veins (the latter sometimes congested and sometimes not, the blood generally fluid). When aconitin was administered subcutaneously to these animals, there was no inflammatory appearance in either the stomach or bowels.

In the case of Dr. Carl Meyer, who died in five hours after swallowing 6/100 of a grain (0.04 gm.) of nitrate of aconitin, the surface of the skin had a white-marble paleness, and the pupils were moderately dilated; the color of the large intestine was pale; the duodenum was much congested, this being more marked towards the stomach; the mucosa of the stomach was strongly hyperemic, having an intense red color; the spleen was enlarged, and full of much dark blood; the liver and kidneys were deeply congested; the lungs, also, were congested; the right ventricle of the heart was distended with blood, and the pericardium contained bloody serum; the brain was generally blood-red; in the cerebral hemispheres were several large circumscribed subarachnoid extravasations; the brain mass showed many red, bloody spots. In a case, recorded by Taylor, in which a man died in three hours after eating a small quantity of aconite root, the only abnormal appearance was a slight reddish-brown patch, the size of a half crown, on the cardiac end of the stomach; all the other organs were reported healthy.

The autopsy, held by Medical Examiner Holden of Massachusetts (case 1, 1894), covered the following appearances, as reported by him: Muscles, normal in color; veins in omentum very prominent; lungs collapsed, no adhesions; hypostatic congestion; heart, right side distended with black fluid blood, and small, soft clots, left ventricle empty; liver, spleen, and kidneys congested; stomach contained one ounce of reddish-yellow frothy fluid, in which much aconite was found by Prof. E. S. Wood; mucosa deeply pigmented; intestine, upper portion, also showed pigmented coloration, diminishing downward along its length; brain and its coverings pale, and its veins filled with blood.

660. Detection.—The detection of aconite in cases of poisoning depends upon the discovery by the microscope, of portions of the plant, if such were taken; upon the isolation of the alkaloid aconitin, on getting the peculiar physiological effect of numbness and tingling sensation upon the lips, tongue, and skin, when a portion of the extract is applied to these surfaces; and, when a sufficiently large amount of poison is present, on getting the complete physiological effect, by administering a portion of the isolated alkaloid to an animal.

Concentrated sulphuric acid dissolves aconitin, forming, immediately, a yellow-colored solution, which, if a considerable amount of alkaloid is present, soon changes to a brown, and gradually passes through a reddish-brown to a violet, then to a colorless solution, in twenty-four hours. This test is delicate for 0.0007 gram.

Phospho-molybdic acid precipitates aconitin from very dilute solutions, in the form of a gray precipitate, which becomes bluish after a while; tincture of iodin, gold chlorid, tannic acid, and Mayer's reagent, precipitate it from a moderate, dilute solution; and corrosive sublimate, potassium bichromate, picric acid, and sulphocyanate of potassium, from concentrated solutions.[11] The principle reliance, however, must be placed upon the physiological test, 0.0006 gram sufficing to produce peculiar sensations when applied to the skin, 0.0007 gram when applied to the tongue; 0.0002 gram will poison a mouse with characteristic symptoms, 0.0007 a small bird; 0.00007 will cause tingling and numbness on top of the tongue; and 0.0007, dissolved in spirit and rubbed into the skin, will cause loss of feeling, lasting for some time.[12]

The isolation of aconitin from organic mixtures is best accomplished by Dragendorff's method.[13] If we are tolerably certain, from the symptoms or history of the case, that aconite or aconitin is the cause of the poisoning, the process can be shortened by only shaking the acid solution with naphtha to remove certain impurities, then, after removing the naphtha, rendering alkaline and shaking quickly with benzol, which can be decanted, after it has separated from the water, into several watch glasses and allowed to evaporate; the residue in one watch glass should be used for the sulphuric acid test; that in another should be dissolved in a drop or two of very dilute sulphuric acid (1 : 50) and the aconitin precipitated by phospho-molybdic acid; that in the third should be dissolved in the same way and treated with a drop of platinic chlorid, which will not precipitate aconitin, but will precipitate a large number of other alkaloids; and that in a fourth should be dissolved in dilute acetic acid, for the performance of the physiological test.

Aconitin resists decomposition of the animal tissues for quite a long period, and has been detected by Dragendorff, in the blood, one month and twenty-three days after death, and in the stomach, two months and nine days after death. It can be isolated from all of the tissues and fluids, and has been detected, not only in the contents of

[11] Dragendorff, op. cit. p. 202.
[12] Lancet, March, 1856, 343.
[13] See § 773.

the stomach and intestines, but in the urine, feces, liver, blood, and kidneys. The remarkable symptoms occasioned by the plant, and the discovery of a portion of that which has been taken, will generally be sufficient to explain the cause of death. The only case in which it was criminally administered is that which is related by Dr. Geoghegan, where the deceased had eaten for his dinner some greens dressed by the prisoner. The latter was convicted upon the medical and the general evidence, no trace of the poison having been discovered in the body. He confessed, before his execution, that the powdered root of aconite had been mixed with pepper, and sprinkled over the greens.[14]

II. Atropin; hyoscyamin; hyoscin.

661. In general.—Under this heading we include the associated alkaloids,—hyoscyamin and hyoscin. These alkaloids are often called the "mydriatics," as one of their characteristic reactions is the dilatation of the pupil of the eye.

662. Properties in general.—These alkaloids, often called "tropins," are, chemically, a tropin in combination with certain tropic acids. Tropin is a pyridin compound, while tropic acid is an aromatic acid. Therefore these alkaloids are saponifiable into tropin and a hydroxy acid. The tropin from atropin is closely allied to ecgonin, which is the nucleus of cocain.

663. Occurrence and properties of atropin.—Atropin is found in Atropa belladonna, commonly known as the deadly nightshade. It crystallizes into needles, having a disagreeable, bitter taste, which are only slightly soluble in cold water, quite so in boiling water, easily soluble in chloroform and warm ether, and somewhat soluble in benzol. It is very soluble in ethyl alcohol and amyl alcohol.

664. Properties of homatropin.—Homatropin is an artificial alkaloid similar to atropin in many of its actions, but much less poisonous, and is used in ophthalmic practice. It is crystalline, slightly soluble in water, easily soluble in ether and chloroform.

665. Properties of hyoscyamin.—Hyoscyamin is isomeric with atropin from some solvents such as benzol and chloroform. It can be obtained in crystalline form; but it separates from amyl alcohol generally as an amorphous body. It is only slightly soluble in cold water, more readily in hot water, soluble in alcohol, ether, chloroform, benzol, and amyl alcohol.

666. Properties of hyoscin.—Hyoscin was formerly supposed to be

[14] Dublin Jour. July, 1841.

an isomer of atropin, but it is probably slightly different, as its most accepted formula is $C_{17}H_{21}NO_4$. It is an amorphous base; but its salts are generally crystalline. It is only slightly soluble in water, but soluble in alcohol, chloroform, ether, and benzol; and this last solvent extracts it easily from alkaline solutions.

667. **Occurrence of atropin and hyoscyamin in belladonna.** — The seeds of the Atropa belladonna are very small, kidney-shaped, weighing about ninety seeds to the grain, and are covered closely with small, round projections. These seeds can be easily identified by an expert, who should have at hand samples of different poisonous seeds for comparison. As belladonna owes its poisonous properties to the alkaloid atropin, it is usual to refer to the alkaloid to demonstrate the peculiar characteristics of the poisonous properties of the plant. Atropin, which exists in the plant combined with malic acid, is generally associated with hyoscyamin, both in belladonna and datura plants.[15] According to W. Schütte,[16] it appears that the younger roots of the wild belladonna contain hyoscyamin only, while the larger roots contain atropin, the latter in small proportion. We have observed that the same rule applies to the cultivated belladonna. The yellow leaves of the wild and black-fruited Atropa belladonna contain hyoscyamin and atropin, the latter in small quantities only.

668. **Occurrence of atropin and hyoscyamin in Datura stramonium.** —The Datura stramonium, fresh and old seeds, contains, chiefly, hyoscyamin, with small quantities of atropin and scopolamin. Mr. Blyth[17] calls attention to the fact that in India the seeds of the white datura have been mistaken for those of capsicum. Datura and belladonna contain the same alkaloid; the seeds of the former should be recognized by the expert when mixed with capsicum seeds. These seeds could be easily distinguished by the difference in taste, the datura seeds having a very feeble, bitter taste, while the seeds of the capsicum are sharp and pungent, and irritating to the mucous membrane. The infusion of the bruised seeds of the datura will have the effect of dilating the pupil, when placed in the eye.

669. **Preparations.**—Belladonna leaves, from which the preparation alcoholic extract of belladonna leaves (U. S. P.) is made; tincture of the leaves of belladonna (U. S. P.); fluid extract of belladonna root (U. S. P.); belladonna ointment, made from the alcoholic extract of belladonna leaves, 10 parts to 100 parts (U. S. P.); bella-

[15] Ber. der Deutsch. Chem. Ges. Bd. 13.
[16] Abstract of the Jour. Chem. Soc. Feb. 1892, 231.
[17] Op. Cit. p. 370.

donna plaster, also made from alcoholic extract of belladonna leaves, 200 parts to 1,000 parts (U. S. P.).

670. Statistics of poisoning.—Mr. Blyth, in his mortality table, to which we have often referred, gives a total of 76 deaths caused by poisoning from belladonna, and three from atropin; out of this total of 79 deaths, 58 were caused by accidents or negligence, and 20 were suicide. There are no deaths reported by the medical examiners of Massachusetts, during the twenty-six years ending 1903, from atropin or belladonna, except in a few instances where belladonna, used in a pill with strychnin and as a cathartic, was taken by mistake by children, playing with the bottle or box containing the same. In these cases the amount of extract of belladonna was only 1/5 of a grain, and strychnin 1/12 of a grain, and the symptoms of strychnin poisoning predominated over those of belladonna.

On the authority of Mr. Blyth, we learn that in Europe and America criminal poisoning from atropin is very rare, while in India it is very prevalent. He states that in 120 cases, recorded in papers and works on Indian toxicology, 63 per cent of the cases were criminal, 19 per cent suicidal, and 18 per cent accidental. He further remarks that the Hindoos, from centuries of practice, have attained such dexterity in the use of the datura as to raise that kind of poisoning to an art, so that Dr. Chevers, in his Medical Jurisprudence for India, declares that "there appears to be no drug known in the present day, which represents, in its effects, so close an approach to the system of slow poisoning believed by many to have been practised in the Middle Ages, as does the datura." Poisoning by atropin and the other mydriatics classed with it will cause so nearly the same symptoms that it is well to include all this class of alkaloids as producing the same effect. The leading symptoms are marked dryness of the mouth and throat; redness of the tongue; thirst; difficulty in swallowing; pupils dilated, with impaired vision and loss of eye accommodation; hallucination with delirious symptoms; inco-ordinate muscular movements, sometimes with convulsions; nausea and vomiting more occasionally occur.

671. Symptoms of poisoning by atropin and its related alkaloids, in general.—The symptoms of acute poisoning by atropin and its related alkaloids will be treated under the same head; we refer to the plants belladonna and hyoscyamus; stramonium, and duboisia myoporoides possess practically the same active principle of atropa and hyoscyamus, which is variously represented by the following names: Daturin, hyoscyamin, and duboisin. The symptoms are:—

1. A hot and dry sensation experienced in the mouth and throat, with thirst, and difficulty in swallowing caused by slight paralysis of the muscles of deglutition.

2. Nausea and vomiting sometimes occur,—especially when portions of the plant have been swallowed.

3. The pupils become dilated and the vision indistinct, due to paralysis of the sphincter iridis and the ciliary muscles. The pupils are so dilated that the iris is sometimes reduced to a very narrow ring.

4. The flush on the face is very marked, the eyes appear brilliant, the pulse is usually small, thin, and rapid.

5. Dizziness then follows, accompanied with restlessness, and great excitement approaching delirium. This delirium is a peculiar form, because attended with visual hallucinations and illusions, the patient frequently imitating these dreams; as, for example, that of a person employed in sewing, or one cutting cloth into strips or picking up substances. This imitative process, as represented by hallucination, is very persistent.

6. Drowsiness with sleep generally supervenes; but, in graver cases, convulsions may appear.

7. An erythematous rash, which resembles that of scarlet fever, often appears on the chest and other parts of the body; the bodily temperature is slightly elevated 2° or 3° F. These latter symptoms may occasionally lead the observer to suppose he has a case of scarlet fever, when, in reality, they are the effects of atropin poisoning.

Some individuals are so susceptible to the action of atropin that, even with very small doses, the throat symptoms may be very prominent; these symptoms have followed the dropping of a solution of atropin into the eye by an ophthalmic surgeon; even when this solution, in which was ½ to ⅛ of a grain of atropin, was dropped into the eyes of a boy, aged twelve, in ten installations during forty-eight hours, at the close of that period the symptoms remarked were a staggering, unsteady gate, dryness of the throat and tongue, flushed face, pupils somewhat dilated, picking at imaginary objects in the air, incoherent muttering on reviving, and occasionally laughing outright to himself; when addressed, he would recover from these hallucinations for a few minutes, then they would return. A few hours later, tetanic spasms came on, with delirium. Complete recovery occurred in a few days.[18]

Three or four drops of a solution of atropin, containing ⅔ of a

[18] London Lancet, 1890.

grain to 1 ounce of water acidulated with acetic acid, were put into the eye of a man with double cataract. In half an hour he had vertigo, and shortly after, all the symptoms of poisoning with belladonna, flushed face, dilated pupils, and incessant hallucinations. His bladder became distended, and he was unable to empty it. Violent delirium continued during the night. He recovered in four days, these unpleasant symptoms having gradually disappeared.[19] Dr. Bethune has reported a case, in which a few drops of a solution of 2 grains of atropin to 1 dram were applied three times in one morning to the eye. In the afternoon the patient was attacked with delirium, and had an uncertain gait, sleeplessness, and difficulty of swallowing. On the next day but one he had another attack of delirium, which somewhat resembled delirium tremens, as he saw imaginary persons in the room.[20]

A similar case is reported by Purley,[21] following the installation of 2/15 of a grain of homatropin (an artificial alkaloid whose physiological effects are less prolonged), instilled into the eyes of a child of seven years, at four different times during one hour; complete recovery occurred after some days.

Symptoms of atropin poisoning may also occur if a belladonna plaster is applied to the broken surface of the skin, or where it is irritated, inflamed, or blistered. Caution should be observed in the application of a belladonna plaster to a portion of the skin where some cutaneous vesicant has disturbed the normal condition of the skin, even if it is only of an inflammatory character.

About one sixth of a grain of atropin is capable of producing unpleasant symptoms. A young man poisoned himself with 2 grains of this alkaloid. No trace of the poison could be detected in the stomach or intestines. Dr. Andrew, of the Royal Infirmary, Edinburgh, had a patient who was treated by atropin, locally, to the eye. She swallowed, one morning, by mistake, between 5 and 6 drams of the solution, containing about two thirds of a grain of atropin. She was immediately sensible of her error, her eyesight failed her, as well as her voice, the muscles of her face were convulsively moved, and she complained of a burning heat in her throat and stomach. She was very restless, and unable to stand. Although emetics and purgatives were given to her, the next day she was violently agitated, and presented the symptoms of a person with delirium tremens. She

[19] Am. Jour. of Med. Sci. (from Gaz. des Hôpitaux) Oct. 1853. p. 540.

[20] Boston Med. and Surg. Jour. April, 1857, p. 201.

[21] New York Medical News.

recovered, but had double vision, spectral illusions, and various disturbances of the nervous system for a week or two.[22]

Morphin and atropin, each given in fatal doses, do not antagonize each other so far as the fatal results are concerned; but a fatal dose of morphin may influence the above-described symptoms, in some cases the symptoms of morphinism predominating, while in others atropinism may be most prominent; for instance, the pupil may be contracted by the opium, or may be dilated by the atropin, yet it must not be forgotten that, just prior to death from morphin alone, the pupil is often dilated, and not contracted; so, too, the effect of increasing the frequency of breathing, from the predominance of atropin poisoning, may obscure the diminution of these movements, which is peculiar to morphin poisoning. The elimination of atropin is by means of the urine; a few drops of the latter may be used as a test by introducing them into the eye of an animal, when the pupil will be dilated, if atropin be present. A very characteristic symptom of poisoning by atropin has been mentioned, *viz.*, paralysis of accommodation of the eye, by which is meant the inability to change the focus of its lens, so that the eye cannot accommodate itself to near or remote objects. This paralysis of accommodation lasts as long as the pupil remains dilated, and may be produced by local application of weak solutions of atropin, and this local paralysis will also occur if general absorption of a sufficiently large dose has taken place.

672. Symptoms of poisoning arising from use of belladonna.—The root, leaves, and fruit of this plant are all poisonous. The berries are black, and have often been eaten by children, in ignorance of their poisonous properties. Dr. Taylor states that they were, on one occasion, openly sold in the streets of London as an edible fruit. Two persons, who had eaten of them, died; and the man who sold them was tried and convicted of manslaughter. A case, graphically described by this author, will suffice as an illustration of the symptoms produced: "A boy, aged fourteen, ate, soon after breakfast, about thirty of the berries, which he had bought in the street. In about three hours it appeared to him as if his face was swollen, his throat became hot and dry, vision impaired, objects appeared double, and they seemed to revolve and run backwards. His hands and face were flushed, and his eyelids tumid; there were occasional flashes of light before his eyes. He tried to eat, but could not swallow, on account of the state of his throat. In endeavoring to walk

[22] Edin. Month. Jan. 1852.

home, he stumbled and staggered; and he felt giddy whenever he attempted to raise his head. His parents thought him intoxicated; he was incoherent, frequently counted his money, and did not know the silver from the copper coin. His eyes had a fixed, brilliant, and dazzling gaze; he could neither hear nor speak plainly, and there was great thirst; he caught at imaginary objects in the air, and seemed to have lost all knowledge of distance; his fingers were in constant motion; there was headache, but neither vomiting nor purging. He attempted to get out of bed, with a reeling, drunken motion; his speech was thick and indistinct. The pupils were so strongly dilated that there was merely a ring of iris, and the eyes were insensible to light; the eyelids did not close when the hand was passed suddenly before them; he had evidently lost the power of vision, although he stared fixedly at objects, as if he saw them. The nerves of common sensation were unaffected. When placed on his legs he could not stand. The pulse was 90, feeble, and compressible; his mouth was in constant motion, as if he were eating something. His bladder was full of urine, on admission. He continued in this state for two days, being occasionally conscious. When, by a free evacuation of the bowels, some small seeds were passed these were examined, and identified as the seeds of belladonna. The boy gradually recovered, and left the hospital on the sixth day after his admission."[23] Total blindness is not an unusual effect of this poison. It was observed in a child, seven years of age, found wandering in the streets of London.[24] Sometimes the cerebral symptoms are much more aggravated than in the above case, there being, frequently, delirium or stupor, which, as well as the other symptoms, is slow in passing off. In a case related in the Lancet, a lady was given, by mistake, a dram of the extract in soap liniment; she fell into a comatose condition in half an hour; the pupils were widely dilated, the hands and feet cold, and the pulse scarcely perceptible. Her jaws were rigid, and there was no vomiting; the stomach pump was used, and she recovered gradually in a few days. Another instance in which it caused serious symptoms, from having been put into soup instead of caramel, is mentioned in the Annales d' Hygiène. The toxical effects of belladonna are produced, when applied locally, or introduced into the rectum. A lady, suffering from hypogastric pain, applied to the abdomen a liniment composed of camphorated oil and extract of belladonna. Forty-eight hours after commencing its use she was seized with delirium. The pupils be-

[23] On Poisons, Am. ed. 1875, p. 725. [24] Lancet, Dec. 1859, p. 561.

came dilated, and there were irregular movements, lipothymy, redness of the face, and a fixed stare. The menses flowed abundantly, anticipating their proper epoch by ten or twelve days.[25] A fatal case of the employment of an enema of the decoction of the root is recorded.[26] The extract varies very much in strength, and is sometimes quite inert.[27]

The disturbance of vision, described in the above paragraph, is a symptom peculiar to a class of poisons and drugs called mydriatics, such as hyoscyamus, datura, and duboisia. This, and all other symptoms peculiar to belladonna, are attributable to its active principle called "atropin;" and the intensity of the symptoms caused by the preparations of the plant, as well as the plant itself, will depend upon the proportion of this alkaloid present in them.

673. Symptoms of poisoning arising from use of Datura stramonium.—Datura stramonium (Jamestown weed) should be classed with Atropa belladonna, as the alkaloid which exists in this plant, and formerly called "datura," has been proved to be identical with atropin, both chemically and physiologically.

Poisoning by this plant is usually accidental, and frequently due to the ingestion of the seeds by children. It is a very common plant in this country, being often cultivated in the garden, and is a household remedy for asthma.

All parts of the plant are poisonous; but the seeds and the leaves are most frequently employed. In some countries it has been, and is still, used for the purpose of producing intoxication with unconsciousness, in order to facilitate the perpetration of criminal designs. It has thus been given, infused in wine or mixed with food. Poisoning by other species of datura is very common in India. During the year 1848, there were treated for it, at the Native Hospital in Bombay, forty-nine males and two females. The powdered seeds are there employed, concealed in rice or other grain. In many cases three stages of symptoms are observed,—delirium, sopor, and coma; in others, only delirium is observed. The primary delirium may be vociferous or merely garrulous, the patient usually manifesting excessive timidity. In both this and the soporific stage, he is constantly engaged in picking at real or imaginary objects, and sometimes in performing such antics as to render laughter unavoidable even on the part of friends. Dr. J. G. Johnson reports the case of

[25] Times and Gaz. Aug. 1859, p. 173.

[26] Casper's Wochenschrift, Feb. 1845, p. 101.

[27] The susceptibility of persons to this poison varies greatly. A dose which would produce little or no disturbance in one individual may cause unpleasant symptoms in another.

a boy in whom the movements were like those observed in chorea.[28] Several of the movements seem to depend upon perverted vision, by which the power of judging of the distance of objects is destroyed, from paralysis of the muscles of accommodation in the eye. Husemann observed a case of poisoning by this plant, in which all black objects appeared green to the patient.[29] In other cases there is complete blindness. The pulse and temperature, although usually natural, undergo, in some cases, extremes of exaltation and depression. On recovery, the person usually recollects nothing since the meal at which he was poisoned, so rapid are its effects.[30] Dr. Duffin, of London, reported the case of his own child, two years old, who died in twenty-four hours after swallowing one hundred seeds, without chewing them. She became fretful, and like a person intoxicated; in the course of an hour, efforts to vomit ensued, together with flushed face, dilated pupils, incoherent talking, and, afterwards, wild spectral illusions and furious delirium; in two and a half hours she lost her voice and the power of swallowing, evidently owing to spasms of the throat. Then croupy breathing and complete coma set in, with violent spasmodic agitation of the limbs, occasional tetanic convulsions, warm perspiration, and a scarcely perceptible, though not frequent, pulse. In other cases the pulse was full and slow, and the general symptoms those of ordinary intoxication, with this remarkable exception of the slowness of the pulse. In a case related by Boerhaave, and in others reported in this country, a scarlet eruption appeared on the face.

Accounts of poisoning, one by the infusion of the leaves, and the other from eating the seeds (which have not a disagreeable taste), may be found,[31] in which great stress is laid upon the eruption, "somewhat like that of urticaria," which appears as one of the toxic symptoms, and varies in intensity with the severity of the symptoms. In females it has produced nymphomania. Kurzak observed priapism of an hour's duration in a case of poisoning with stramonium seeds.[32] Dr. Bobierre, professor of chemistry at Nantes, drank, by mistake, a small quantity of an infusion of the leaves and seeds. In a quarter of an hour he began to feel heavy, and had an uncomfortable feeling of constriction in the neighborhood of the larynx. His pupils were dilated, and the secretion of saliva, perspiration, and

[28] Am. Med. Times, I. 22.
[29] Jour. f. Pharm. II. 191.
[30] Brit. and For. Med-Chir. Rev. Jan. 1851.
[31] Boston Med. and Surg. Jour. Aug. and Oct. 1872, pp. 81, 240.
[32] Schroff, Pharmacol. p. 532.

urine, was entirely suppressed.[33] The external application of the bruised leaves may give rise to the symptoms of poisoning. An overdose of the officinal extract has produced fatal effects.

674. **Active principles of stramonium.**—The active principles are the same as in the belladonna. The alkaloid, which was formerly called daturin, is now known to be identical with atropin. The experiments of Ladenburg[33a] have shown the same two alkaloids in the Datura stramonium which exist in the belladonna, *viz.*, the heavy and light atropin, or, in other words, atropin and hyoscyamin. The chemical tests and analysis are precisely the same as for belladonna.

675. **Action and effect of Hyoscyamus niger.**—Hyoscyamus niger (henbane) also possesses the alkaloid hyoscyamin. It is nearly similar in its action to the alkaloid contained in Atropa belladonna, and should be considered under the same head as the two preceding plants.

Dr. Cabot, of Boston, gave three teaspoonful doses of the tincture of hyoscyamus at intervals of an hour. Ten minutes after the last dose the face began to swell, and became red and polished; the eyes were closed; and the patient was able to speak only with the greatest difficulty, on account of the swelling of the tongue and lips. The red discoloration of the skin extended as far as the navel, and was attended with intolerable itching and burning.[34] Delirium and hallucinations, after 14 grains (0.91 gm.) in divided doses, were met with by Reinbold, of Hanover.[35]

The only two cases of death alleged to have been caused by hyoscyamus were reported, the one in 1715, by Walther,[36] and the other by Lindern, who is quoted at secondhand by Orfila.[37] This result may, therefore, be regarded as extremely rare. A case of poisoning by the fruit of hyoscyamus is recorded in the British and Foreign Med.-Chir. Review (1859), with symptoms almost identical with those in the case of Dr. Cabot, related above.

All parts of this plant are poisonous. The root is long, tapering, whitish, and fleshy, and bears considerable resemblance to parsley and parsnip roots, and has been eaten by mistake for them. Dr. Houlton states that, in a monastery where the roots had been eaten for supper by mistake, the monks who partook of them were seized in the night with the most extraordinary hallucinations, so that the place became like a lunatic sylum. One monk rang the bell for mat-

[33] Jour. de Chim. Méd. 1851, p. 539.
[33a] Berichte der deutsch. chem. Gesellsch. XIII. p. 909.
[34] Am. Jour. of Med. Sci. Oct. 1851.
[35] Casper's Wochenschrift, 1840, No. 8.
[36] Wibmer, Wirkung, etc. III. 149.
[37] Toxicologie, 5 ème éd II. 304.

ins at twelve o'clock at night; of those of the fraternity who attended to the summons, some could not read, some read what was not in the book, and some saw the letters running about the page like so many ants.[38] Orfila relates two cases in which paralysis, delirium, and insensibility, together with tetanic symptoms, were caused in two soldiers who ate of the young shoots of this plant.[39] The seeds are still more active. Two young children, having eaten some of them, became actively delirious, and even maniacal, striking and biting all who came in their way. Their faces were red, hot, and swollen, and the pupils dilated. They were gradually restored by the use of emetics, local depletion, and sinapisms to the extremities.[40] Another similar case is related in the same journal, but, the seeds being unripe, the symptoms were still more alarming.

The action of both hyoscyamus and its alkaloid resembles so closely that of belladonna and atropin that reference is made to these articles (§§ 671, 672); the only marked difference between these two drugs is their action upon the brain; in this respect, hyoscyamus appears to produce a more decided impression than belladonna, and especially produces a quieting effect upon the muscular system; in fact, it has been used without serious consequences in large doses (½ grain to 1 or 2 grains, 0.032 gm. to 0.065 or 0.130 gm.) in insane asylums in England to control voluntary movements.

676. Preparations of Hyoscyamus niger.—The medicinal preparations are the tincture and extract. Both of these vary greatly in strength. The active principle of the drug is represented by its alkaloid, hyoscyamin, the strength of which varies with each lot, because it is not usually prepared in a pure state.

677. Properties and action of duboisin.—Duboisin, an alkaloid from the Duboisia myoporoides, of the order of Solanaceæ, is identical with hyoscyamin. Dr. Norris, of Philadelphia, found that dilatation of the pupil occurred in half the time required after a dose of atropin. Eight one-hundredths of a grain (0.0052 gm.), administered by injection under the skin, was followed by dryness of the throat, and indistinctness of vision, whereas, twice this dose quickened the pulse, flushed the skin, and impaired muscular movements, differing in this respect from the action of atropin; it always differs from atropin by causing placid indifference and inertness, or even faintness, but no drowsiness. It opposes the sweating effects caused by pilocarpin. In one case mentioned by Dr. Norris, an exceedingly minute

[38] Lancet. July 6, 1844.
[39] Toxicol. Gener. II. 264.
[40] Henke's Zeitsch. 1848, 4 H. p. 516.

portion of duboisin sulphate was applied to the surface of the eye, where it melted, and a few moments later the patient became dizzy, her face flushed, and she became wildly delirious, very restless, and picked at surrounding objects. According to Wecker, this alkaloid produces less irritation of the eye's surface than atropin. Another difference is shown by the fact that duboisin acts more promptly in paralyzing the muscles of accommodation in the eye. The medicinal dose of duboisin is stated as 1/60 of a grain. (0.0011 gm.)[41]

678. **Lethal dose.**—Kobert[42] quotes from the statistics of atropin poisoning collected by Falk that recovery occurred in 88.4 per cent of all cases. Peterson and Haines call attention to a case reported by Ott,[43] where the subcutaneous injection of ⅓ of a grain (0.021 gm.) is one of the few instances of a fatal result in the many cases of dangerous symptoms following a medicinal dose of atropin. In a case reported by Massachusetts Medical Examiner Oliver (case 4, 1895), death is reported as due to the injection, by an ignorant practitioner, of ⅛ of a grain (0.008 gm.) of atropin in the arm of a feeble patient, four weeks after childbirth. Though the symptoms of poisoning may appear to be very dangerous at first, recovery is more likely to occur than a fatal issue.

The dose of belladonna necessarily fatal to human life is not definitely known, and will depend upon the amount of atropin actually present in the preparation under suspicion. According to Winckler,[43a] the proportion of alkaloid present in the root of belladonna is about .5 per cent, and, in the leaves, it varies from 0.41 to 0.49 per cent; according to Flückiger and Hanbury,[44] the dried leaves yield 0.44 to 0.48 per cent of atropin, and the young roots contain larger proportions of atropin than the old roots, the maximum proportion in the former being 0.60 per cent, and in the latter 0.25 to 0.31 per cent; the alkaloid resides chiefly in the bark. "Manufacturers of atropin employ the root exclusively."

679. **Treatment.**—The removing of the contents of the stomach, if the poison has been swallowed; perhaps, the introduction of a compound solution of iodin, 6 drops diluted with water, 3 or 4 ounces, and the stomach immediately washed out; or, tannic acid can be used in solution in a large amount of water, and then pumped

[41] The National Dispensatory, Stillé's and Maisch, Phila. 1879. p. 517.

[42] Lehr. die Intoxikationen, p. 606, Stuttgart, 1903.

[43] Medical News, Phila. 1895, Vol. LXVII. p. 628.

[43a] Pharm. Jour. 1872, 1029.

[44] Pharmakographia, London, 1874, p. 412.

out. The object of administering these two substances in the stomach would be to precipitate the alkaloid in an insoluble state. Charcoal powder could also be introduced, which would collect the alkaloid, and then could be washed out of the stomach. Pilocarpin is a physiological antidote to atropin. Morphin is an antidote only in a symptomatic sense, and, therefore, should be used very cautiously to counteract the effects of atropin. The dropping of physostigmin into the eye is also recommended by Kobert as an antidote. The use of the ice cap to the head would serve to counteract the mental cerebral disturbances.

680. Post-mortem appearances.—The post-mortem appearances produced by belladonna and atropin are by no means characteristic. Most generally there is a marked redness of the lungs, of the meningeal membranes of the brain, and of the retina; there has been observed, also, in animals, hyperemia at the base of the brain, in the choroid plexus, and in the lateral ventricles. Congestion of the retina is especially noticed after slow or repeated poisoning. In every case this state of hyperemia is only the first degree of interstitial hemorrhage which is met with quite frequently. (Tardieu.)

The diffuse redness of the gastric mucous membrane should be ascribed to the brandy which has been used an a vehicle for the poison. (*Ibid.*) Rosenberger[45] relates a case in which the brain mass, the cerebellum, and the spinal cord were the seat of numerous capillary apoplexies. Kobert[46] states that these are of little value; of course, dilatation of the pupils is an important symptom of poisoning by a mydriatic. It should be said, however, that in this respect the post-mortem symptom may not necessarily be due to a medical mydriatic. Vaughan and Novy refer[47] to a case where Haines and Vaughan were appointed a commission to inquire into the truth of the tests "obtained by a toxicologist who had reported 4 grains of atropin in the stomach of a man who had been dead some weeks." The chief test relied upon by the chemist was that an ounce of the extract made from the man's stomach dilated a cat's pupil as much as a solution of 4 grains per ounce of atropin sulphate. It is needless to comment on the validity of such evidence. These authors, in the same paragraph, refer to certain putrefactive products which resemble, in their mydriatic properties, atropin and hyoscyamin. These substances are known in physiological chemistry as homat-

[45] Canstatt's Jahresbericht, 1845, p. 295.

[46] Kobert, op. cit. pp. 33, 134.

[47] Cellular Toxins, 1902; Lea Bros. & Co. Phila. p. 244.

ropins, and are found in decomposing fish, corned beef, putrid game, and poisonous sausage. The symptoms of poisoning resemble very closely those from belladonna. If berries of the deadly-nightshade have been swallowed, and are found in the stomach, the mucous surface would be stained a dark blue color. In case datura was swallowed, there would be noticed extravasation and inflammation of the mucous lining of the gastrointestinal tract. As before remarked, in the case of atropin being the cause of death, enough of it would be found unchanged in the urine, in the brain, and in the liver of the cadaver, to be used as a test on the eye of an animal, to develop the dilatation of its pupil.

681. Chemical examination.—The evidences of poisoning by belladonna depend upon the detection of portions of the plant, such as the seeds and leaves, when such have been taken, in the vomitus and feces; and upon the isolation and recognition of the active principle atropin in the vomitus or urine during life, or in the tissues and blood after death.

The principal tests for atropin are the peculiar crystalline precipitates produced in solutions of its salts by a solution of bromin in hydrobromic acid, by picric acid, and by trichlorid of gold.[48] One of the most delicate precipitating agents for atropin is Mayer's reagent, which gives a precipitate in solutions containing only 1 part of atropin in 7,000 parts of solution; and this reagent has been recommended by Dragendorff for the quantitative estimation of this alkaloid. Bichlorid of platinum gives no precipitate with solutions of the salts of atropin as it does with most alkaloids; atropin is also precipitated from its solutions by phospho-molybdic acid, corrosive sublimate, tannic acid, potassium bichromate after a while, and a solution of iodin in iodid of potassium, if the solutions are not too dilute.

To a drop of an aqueous solution of a salt of atropin, add a drop of a hydrobromic acid solution saturated with bromin. Atropin or hyoscyamin produces a characteristic crystalline deposit, which can be compared with a test of a solution of a known atropin salt. Hyoscin produces a precipitate, which is generally quite different from those obtained by atropin or hyoscyamin.[49]

A drop or two of a solution of an atropin salt other than the chlorid is moistened with a drop of fuming nitric acid, and evaporated to dryness on the water bath. The residue is then moistened

[48] See Wormley, Micro-chemistry of Poisons.

[49] Wormley, Micro-chemistry of Poisons, 2d ed. p. 642.

with a few drops of freshly prepared alcoholic potash solution. The substance then acquires a fine violet color, soon turning to dark red and disappearing. This reaction is sensitive to 0.000001 gram with pure salts.[50]

The best test is, however, the physiological one, which depends upon the readiness with which a solution of atropin, introduced into the eye, dilates the pupil; in performing this test, comparative experiments should always be made, the animal being placed in the same light and kept there, the width of the pupils measured, and a drop or two of the suspected fluid introduced into one eye; in a very short time, if atropin be present, the pupil of that eye will become dilated, while that of the other eye will remain unchanged. If atropin be introduced into the eye of an animal, the pupil begins to dilate in one or two minutes; if it be injected subcutaneously, in from five to ten minutes; and if swallowed, in from twenty to thirty minutes.

If poisoning has resulted from the ingestion of the berries, as is very often the case in children, the seeds can almost always be detected in the contents of the stomach or intestines. These may be recognized by their kidney shape; the horseshoe-shaped embryo, which is situated in the centre of the seed; their gray color; their size, which is about 2 millimeters long by 1½ broad; and their rough, uneven surface.

The coloring matter of the berry is also important, and can usually be seen in the contents of the stomach or vomitus; and its presence is additional confirmatory evidence, when the seeds have been detected. A substance having a blue fluorescence is also present in the belladonna, and exists in very large amount in the berry; this is soluble in acid fluids, from which it can be removed by rendering alkaline with ammonia and shaking with amyl alcohol; and from the amyl alcohol it can be removed by shaking with acidulated water.

In order to isolate atropin from organic mixtures, the same process may be used as has been recommended in the case of opium and morphin (see § 773), the atropin being removed from the ammoniacal fluid by shaking it with benzol, care being taken to keep the fluid warm, while extracting with benzol, on account of the liability of the atropin to separate from cold benzol into crystalline form.

Atropin withstands decomposition of the animal tissues for a long time, and was detected in a mixture of organic substances which had been decomposing for two and a half months. (Dragendorff.)

[50] Vitalli, Bruxelles, 1880.

One of the most important sources of the elimination of atropin is the urine, in which it may be detected even after it has been completely absorbed from the stomach, or after introduction hypodermically; so that this fluid should always be carefully examined in all cases of suspected atropin poisoning.

III. Cocain.

682. Occurrence and properties.—Cocain is the alkaloid obtained from the leaf of Erythroxylon coca, a plant indigenous to South America. It is used widely in medicine and minor surgery. The free alkaloid crystallizes in prisms (m. p. 98°), dissolves slightly in cold water, easily soluble in alcohol, ether, and benzol. Chloroform, methyl alcohol, and ligroin dissolve it when hot; but crystals of the alkaloids separate easily on cooling. It is easily decomposed into methyl alcohol and benzoyl-ecgonin; this decomposition takes place in the living body, and ecgonin may be found in the urine.

Cocain is used in medicine in the form of its hydrochlorid salt, which crystallizes in prisms or scales (m. p. 181.5°), and is freely soluble in water, quite soluble in chloroform, almost insoluble in alcohol, ether, benzol, and ligroin.

683. Preparations.—The leaves of the Erythroxylon coca are used to make fluid extract of coca (U. S. P.), which contains 1000 grams of the powdered leaves (No. 40 mesh) in 1000 cubic centimeters of diluted alcohol, and represents from 1/5 to 1 per cent of the alkaloid cocain. The other preparation in the pharmacopœia (U. S. P.) is the cocain hydrochlorate, from which alkaloid cinnamyl-cocain and other bases are excluded. As cocain possesses the poisonous properties of the plant itself, we shall confine our description to its poisonous action as representing that of the plant itself.

684. Symptoms in general.—The symptoms of cocain poisoning in man vary in different persons. It may generally be said that small quantities of the alkaloid produce excitement of the mind, which may be either agreeable or disagreeable to the subject; the mental condition is somewhat anxious and confused. On the other hand, a small dose may produce a state of malaise which somewhat resembles the symptoms following small doses of morphin, but not necessarily, like the latter, inducing sleep. Cardiac pulsations are quickened, respiratory movements are also more frequent than normal, the pupil of the eye is dilated, and headache with the peculiar sense of dryness in the throat previously described in atropin poi-

soning is experienced. Like morphin, after small doses the response to the irritation of the skin is more active, showing an excitation of the reflex action, which may be represented by muscular tremblings of a slight convulsive character. At a later period, more general convulsions of groups of muscles may appear, with an exhilaration of the heart's impulses, more rapid and difficult breathing, which may end in a convulsion. On the other hand, the convulsive effects of the poison may be absent, and the patient may pass into a faintness and collapse, with cold and cyanotic skin, accompanied by slow and weak action of the heart. These last symptoms may end in a gradual collapse and cessation of life.

685. Local action.—The local action of cocain upon most parts of the body is that of an anesthetic, which is apparently due to a paralysis of the terminal ends of the sensory nerves, excepting those which convey a feeling of heat and cold. For instance, application of cocain to the tongue disguises the taste of bitter substances, and also destroys, in part, the sensation to sweet and acid fluids, yet salt is recognized as in normal conditions; application to the local mucous membrane of the nose destroys the sense of smell entirely. For this reason the local application of cocain to the mucous membranes in the interior of the body induces local insensibility to pain and to touch, that is, if the cocain is applied in sufficient concentration. Applied to the surface of the unbroken skin, this abolition of the sensibility is but feeble, because the absorption through the horny surface of the skin is accomplished very slowly; on the broken skin, where the epidermis is removed or abraded by vesicants or by skin disease, the abolition of local sensation may be produced very much the same as when applied to the interior mucous membrane. Subcutaneous injection produces insensibility wherever the solution attacks the terminals of the sensory nerves. When injected into direct contact with a nerve trunk, the function of that nerve is suspended, and even that of the organ which that nerve innervates; when injected into the spinal cord or its canal, general anesthesia is produced in the portion of the body innervated from the nerve center which is bathed in the solution. The ingestion of a solution of cocain by the mouth may not necessarily be followed by a general anesthesia, probably because there is not sufficient quantity in the solution in contact at any one time with the terminal ends of the nerves which convey sensory reflex action.

Besides the loss of sensation following the local application of cocain to a mucous membrane, there is a distinct feeling of constric-

tion of the throat or passage, due to the contraction of its capillary blood vessels, which can be noticed in the distinct paleness of the surface of the mucous membrane. The abolition of sensation following the subcutaneous administration is transitory, and disappears when the poison has been absorbed into the general system.

686. **Action when applied to surface of eye.**—The application of solutions of cocain to the surface of the eye produces in that organ, also, abolition of sensation to pain and touch, and a contraction of the capillary vessels in the conjunctiva. This is followed by dilatation of the pupil with partial loss of the muscular power of accommodation; this dilatation is much less than that produced by atropin; the reflex response to light is also unaffected, *viz.*, the pupil contracting in strong light and dilating in the dark. The vessels of the iris share in the reduction of the caliber of its capillary blood vessels, which gives the appearance of an extended whiteness to the sclerotic surface, apparently pushing forward the globe of the eye, and reducing the interior tension of the eye ball. These same symptoms, in a modified form, follow the injection of cocain in the neighborhood of the eye. In birds, cocain does not cause dilatation of the pupil.

687. **Symptoms of chronic poisoning.**—The use of cocain as a substitute for morphin eating has rapidly become a serious question in therapeutics. The substitution of cocain for morphin in cases of chronic morphinism does not result in relieving the craving for the use and effects of both these alkaloids. Symptoms of cocainism are: Disturbances of digestion, loss of appetite, increase of salivary secretion, as primary effects; afterwards, sleeplessness, muscular tremblings, hallucinations, convulsions of groups of muscles, generally followed by insanity and delirium; these symptoms, accompanied with disturbed muscular action and with loss of sensation, are the effects of its long-continued use.

Absolute discontinuance of the drug will cure these symptoms, if they have not proceeded too far, and without any physical depression; though there is much mental disturbance and a strong feeling of restlessness, which prevents the patient himself from enforcing abstinence in the use of the drug.

688. **Its use in the production of surgical anesthesia.**—In addition to the transitory effects of its local anesthetic properties for surgery upon the eye, cocain has been introduced to procure anesthesia in the more serious and prolonged surgical operations, so as to almost rival the anesthesia of ether or chloroform. At first, Reclus used strong solutions, injected to prepare the way for the knife, each step for-

ward in the operation being preceded by successive injections of cocain to abolish sensation in the layer of tissues into which the knife was to be introduced. This is so serious a method, requiring dangerous doses of the drug, that it is now rarely used. Schleich introduced, under the name of "infiltration anesthesia," a more satisfactory method of accomplishing local anesthesia for surgical practice. By this method 0.1 per cent of cocain and 0.8 per cent of sodium chlorid were introduced under the tissues by means of a fine subcutaneous syringe with very light pressure; the structure surrounding the needle puncture became swollen and edematous, and could be cut into without pain, and, when the tissue was cut, the fluid escaped through the incisions; thus no general absorption occurred, and no symptoms of poisoning arose. Other modifications of the process of an extensive subcutaneous injection of cocain near the nerve supplying the part to be operated on were suggested by Oberst. Absorption by the general system could, in this way, be prevented by the Esmarch ligature being applied above the point of the needle puncture. This method is more suitable for operations upon the extremities.

Bier and Tuffier developed a process, suggested by Corning, of New York, as follows: A long, hollow needle was passed into the spinal canal between the laminæ of the lumbar vertebræ, through which, 1 cubic centimeter of a 2 per cent solution of cocain hydrochlorate was injected, pains being taken to withdraw an equal amount of cerebro-spinal fluid. The actual amount of cocain introduced was ⅓ of a grain (0.02 gm.). After a few moments, numbness followed, generally in the feet at first, but sometimes in the lower part of the trunk, spreading upwards rapidly, until sensibility to pain was lost everywhere below the diaphragm and sometimes in the thorax; in some cases even the head was found to be in a state of anesthesia. The sensations of warmth and cold are less quickly affected, that of touch is conserved to some extent, and the limbs can be moved readily; though the motions are retarded, consciousness remains. This condition lasts from a half hour to an hour, after which sensation gradually returns. Twitchings of the muscles are often seen in the beginning of this anesthesia; but the muscles never relax as they do under ether and chloroform. Headache, nausea, and vomiting occur, generally, during or after the operation. Under this practice the cerebro-spinal fluid contains a large number of polynuclear leucocytes; but this fluid resumes its normal condition only after several days. No fatality has yet been reported, though this cocain anesthesia method has been employed in over 2,000 cases.

689. Lethal dose.—About ⅔ grain (0.04 gm.) of cocain injected subcutaneously caused the death of a woman aged seventy-one years, in five hours (Dixon Mann); 3½ grains (0.23 gm.) injected into the breast of a woman, preparatory to an operation, produced death in twenty-four minutes. The two following and the two preceding cases are related by Dr. Luff: 0.032 gm. of cocain injected into the gums of two adults produced alarming symptoms. 20 grains (1.2 gm.) of cocain in a solution were accidentally swallowed by a man, who died an hour after the administration.[51] In this last case the post-mortem showed the left kidney entirely destroyed, and the right kidney in a tuberculous condition. It should be remarked in this connection that cocain is eliminated rapidly by the kidneys, when in their normal condition; thus, abnormal renal conditions may have increased the danger in this case. Dixon Mann[52] mentions a case in which recovery took place after the ingestion of 46 grains of cocain. It is a striking fact that persons addicted to taking continued doses of morphin can take large quantities of cocain without serious dangerous symptoms.

690. Treatment.—When the blood pressure is at high tension, amyl nitrite has been recommended to reduce this condition; small quantities of chloroform or ether can be inhaled to counteract convulsions. If the poison has been taken by the mouth, the stomach should immediately be evacuated, mechanically, by the use of large quantities of water pumped in and out, or by simple emetics. To stimulate the respiratory movements, cautious use of ammonia vapor has been useful. The most beneficial treatment in cases of threatening asphyxiation would be the inhalations of pure oxygen and artificial respiration.

691. Action, in general.—The action of cocain is twofold, namely, on the central, and on the peripheral, nervous system. A small dose of cocain excites the spinal cord and brain; in large doses, convulsions and paralysis follow. The first abolition of sensation is induced by the action on the peripheral nervous system. Action on the central nervous system interferes with the accommodation of vision, and brings on dilatation of the pupil, and it has also occurred when there is a paralysis of the vagus; this explains the action of the slowing of cardiac pulsations and respiratory movements. Another peculiar action of cocain is shown by the disturbance of the co-ordinate or balancing powers, resulting in an animal moving, generally around

[51] Luff, op. cit. Vol. I. p. 314, and London Lancet, 1889.

[52] *Ibid.* and Forensic Med. Toxicol.

a circle; the symptoms are similar to those which have been described by Professor Bernard, where portions of the cerebrum have been removed. It is held by some authorities that cocain produces action through its power as a protoplasmic poison. It would seem that the center of reflex action situated in the medulla oblongata is inhibited, though its general action as a protoplasmic poison would explain, first, the excitement of the brain, then, of the medulla oblongata, and, finally, of the whole spinal cord, followed, as usual, from exhaustion in such cases by an inhibition of their functions. There seems to be difference in opinion among authorities as to whether these central nervous derangements may be effected within themselves, or by secondary action through the contraction of the blood vessels, due to the stimulation of the vasoconstrictor nerves; sections of the splanchnic nerve seem to relieve the arterial tension. Estimates of the amount of cocain excreted by the kidneys of dogs, in whom this poison is absorbed, show that 95 per cent of the amount ingested is destroyed in the tissues; while, in the rabbit, all of it seems to be destroyed in the tissues. Man is, however, more susceptible to its action than these lower animals.

692. Post-mortem appearances.—The post-mortem appearances are those usually presented in cases of death from asphyxiation; that is, an excess of dark and fluid blood in the lungs, kidneys, liver, and brain, with venous congestion.

693. Tests and detection.—Ecgonin is the base of atropin which is found in cocain. There are no distinctive chemical tests, however, for this alkaloid, and reliance must be placed chiefly upon its physiological tests, and, to a less extent, upon the formation of benzoic acid after saponification.

The physiological test is the sense of numbness and characteristic insensibility to touch, upon the application of cocain to the skin. In strong solution, there is blanching of the mucous membrane; and, if applied to the pupil of the eye, dilatation is noticed, but this is not so marked as in atropin.

Potassium permanganate with the chlorid of cocain gives a violet crystalline precipitate. The presence of impurities may, however, cause the reaction to fail.[53]

A few drops of ferric chlorid added to a solution of salt of cocain produce a yellow solution, which on heating, turns orange and then red.[54] Vitali's test for atropin is applied to a solution containing co-

[53] Giesel. Pharm. Zeitg. 1886, XXXI. [54] Arch. f. Pharm. 1889, 994.
132.

cain, and a peculiar odor resembling that of peppermint is given off, but no red color is produced.[55]

A few drops of gold chlorid solution added to a solution of cocain chlorid form crystals which are more or less stellate in appearance.[56]

Cocain is best separated from organic mixtures by the process described under opium. It is to be found in the residue extracted by ligroin, or better, by benzol, from the akaline fluid.

IV. Coniin.

694. Occurrence and properties.—Coniin is the principal alkaloid that has been isolated from Conium maculatum, commonly known as the poison hemlock. The other alkaloids are conhydrin, pseudo-conhydrin, methyl coniin and ethyl piperidin.

The active principle of common hemlock is a most virulent poison and a local irritant. A single drop of coniin applied to the eye of a rabbit killed it in nine minutes; and, when 2 grains (0.13 gm.) of the muriate of coniin were injected into the femoral vein of a young dog, it died before there was time to note the interval.[57]

Conium is the poison which is generally known to have been the cause of the death of Socrates. As the plant conium resembles in appearance "fool's parsley" (Æthusa Cynapium), it has sometimes been eaten through mistake. The entire plants can be distinguished from each other by the dark spots found on the thicker portions of the spotted hemlock's stems, there being no such spots on the stems of "fool's parsley;" but these spots are not found on stems or branches of hemlock which are smaller than the little finger. In the absence of the thicker portions of the stems, the two plants can be distinguished by the character of the involucels around the flowers. These, in the case of conium, consist of very short bracts, which, at first sight, are scarcely visible; whereas, in "fool's parsley," the involucels consist of long, slender, pendent bracts, which hang down on one side of each little group of flowers. (Luff.)

The spotted hemlock, which is rather a common plant growing in waste places and flowering during June, July, and August, is both fetid and poisonous. Its active properties of poisoning reside in the volatile liquid alkaloid coniin, united with a crystalline alkaloid, conhydrin. The synthetic compound of pure coniin prepared by

[55] Jour. Pharm. et Chim. 1890.
[56] Peterson and Haines. Text-book, L. M. and Tox. 1904.
[57] Christison.

Ladenburg differs from that separated from the plant by having no action on polarized light; but when united with dextrotartaric acid, one portion may be separated by evaporation, which he called "dextro-propyl-piperidin;" this is in every respect identified with coniin made from hemlock.

695. Statistics of poisoning.—According to Falk,[58] out of a total of 17 cases of death, both from coniin and hemlock, collected in medical literature, there was 1 suicide; 1 was caused by local application of the extract to a cancerous breast; 1 by the injection of an infusion of hemlock leaves; and 2 were homicidal; the remaining 12 were cases in which the root, leaves, or other portions of the plant had been ignorantly or accidentally eaten. In the table of the Massachusetts medical examiners, there are 3 cases of suicide reported from taking oil of hemlock. Mr. Blyth's table of mortality contains 4 cases of fatal poisoning by hemlock, due to accident or negligence.

696. Symptoms of poisoning.—The poisonous properties of this plant reside chiefly in the leaves, but exist also in other parts. The accounts of its action upon the human system are somewhat contradictory. Some authors attribute to it positive narcotic properties. Orfila quotes the case of a soldier, who, having eaten of some broth into which hemlock had been put, went to sleep immediately after his supper. A couple of hours later, he was found still lying on the ground in a profound sleep, insensible. His pulse was extremely slow, the extremities cold, the face swollen and livid, and the respiration labored. He died in three hours. Some cases are related in which delirium and fatal convulsions were said to be due to this poison. Other authors state that these symptoms are not produced by coniin, which is supposed to be the active poisonous principle of the plant, nor do they agree with the observations of others,—especially of recent date. Yet many of the inconsistencies occurring in medical literature concerning the action of hemlock preparations are explicable on the ground of the uncertainty of the proportion of the active principle contained in the medicinal preparations. Dr. Pliny Earle tried the effect of the extract of conium upon himself. The preparation seems to have been a feeble one; for, although the dose was steadily increased until it reached 60 grains (3.89 gm.) three times a day, and seven such doses were taken, the effects were by no means striking. No soporific effect, however, resulted; he experienced merely the sensations of fullness of the head and

[58] Prakt. Toxicologie, p. 273.

eyes, a "tendency to vertigo," double vision, and a great feebleness in the limbs.[59]

Dr. Hosea Fountain, however, who prepared for himself an extract from the fruit or seed, and took 12 grains (0.74 gm.) of it, began to experience its effects in half an hour. He had a feeling of lightness in the head, dimness of vision, and muscæ volitantes before his eyes; "very soon," he says, "a numb, pricking sensation was felt in the fingers, extending gradually to the elbows, producing a stiffness of the muscles of the parts, making it difficult to move the forearm and hand. In a few minutes the same sensation was observed in the feet, creeping slowly upward, until it reached the upper part of the thigh. The eyes now began to feel uncomfortable, causing me to brush them frequently, to clear apparent obstructions from the lids. The pulse was soft and feeble, but not more frequent than usual." Having dismounted from his horse, he found so much difficulty in walking that he required assistance, and the lower limbs appeared to be nearly paralyzed. This partial paralysis of the limbs continued throughout the whole day, although the head symptoms disappeared under the influence of tobacco and rest. No soporific effect was produced.[60]

In an undoubted case of death from eating hemlock, the symptoms were very much like those just described. The man's consciousness and intelligence were not affected; but he lost his sight completely, and was unable to walk. He seemed, also, to have lost all muscular power in his arms, and the power of deglutition and speech; several efforts were made to vomit, but they were ineffectual. His pulse and breathing, as well as the heat of the skin, were perfectly natural. Death ensued in three hours after eating the poisonous plants, without convulsions, but apparently from paralysis of the heart and lungs. The lungs are paralyzed long before the heart, so that it is often possible to restore the patient, and to save his life, by prolonged artificial respiration.

Ordinarily, the symptoms caused by coniin, the active principle of hemlock, may be thus described: When the dose taken is large enough to produce the peculiar action of this drug, the person experiences physical languor, impairment of muscular movements, disturbed vision, and dizziness. If he attempts to walk, his limbs feel heavy and numb, and he may totter or fall from muscular weakness of the legs. Quiet with an uncontrollable desire to keep the eyes closed accompanies the other symptoms. The individual may also

[59] Am. Jour. of Med. Sci. July, 1845. [60] Am. Jour. of Med. Sci. Jan. 1846.

suffer from a headache in the temples or forehead, and disagreeable sensations of the throat amounting to nausea or vomiting. The muscles of the eye are incapable of accommodating vision of remote or near objects. The pupil, may, nevertheless, be natural in appearance in spite of this disturbance of vision (Husemann), but sooner or later it may become expanded or dilated. The pulse rate is slow at first, but afterwards becomes rapid. Five fatal cases of poisoning by hemlock are recorded in modern literature.[61]

In short, these symptoms are comprised under the head of nausea with abdominal pains, vomiting, diarrhea, headache, imperfect vision, and dilatation of the pupils. There is a peculiar sense of weight in the legs, with increasing muscular weakness, followed by paralysis spreading upwards to the trunk, the arms being later affected; then, difficult breathing occurs, the respiratory movements gradually becoming slow; difficulty of swallowing, drowsiness followed by convulsions, and coma, sometimes with delirium, occur, if death be delayed. The convulsions are attributed to the general asphyxia caused by the drug. The concentrated free alkaloid coniin is locally a caustic; the effects of the poison are quite rapid, fatal results occurring in from one to three hours.

697. Lethal dose.—Generally speaking, one drop of pure coniin may be a poisonous dose, though none such have been reported. According to Kobert, a little over 2 grains (0.15 gm.) of the pure alkaloid is the lowest fatal dose, though ½ to 1 grain (0.03 to 0.06 gm.) is a dangerous dose. The fresh plant is said to contain from about .04 to .09 per cent, and the fruit about 0.7 per cent of coniin. The tincture of conium is about ⅛ of the strength of the fluid extract of conium (U. S. P.). The medicinal dose of the former is 60 minims (3.68 c.c.); and of the latter, 15 minims (0.92 c.c.) repeated in three hours by a dose of 5 minims (0.3 c.c.).

698. Treatment.—After evacuation and washing of the stomach, the only treatment that has met with any success in animals is in maintaining artificial respiration, or by a subcutaneous injection of .9 gr. (6 mg.) of apomorphin. The bodily temperature should be kept warm by hot wraps, followed by the administration of tannin or gallic acid or strong tea by the stomach; this may precipitate the alkaloid which then can be washed out. Stimulants for increasing the heart's pulsation and the respiratory movements would be advisable.

[61] Husemann, Die Pflanzenstoffe, p. 296; Edin. Med. and Surg. Jour. 1845; The Sanitarian, June, 1875; Amer. Jour. of Med. Sci. Jan. 1846; case of F. W. Walker reported in detail in the New York Times for April 5th and 6th, 1876.

699. Action.—Experiments on animals would seem to show that they are less susceptible to the small doses of coniin which appear to produce poisonous effects in man. Dragendorff experimented on the action of coniin given to five cats in quantities of from 0.8 grains to 8 grains (.05 to .5 gm.); symptoms of dilatation of the pupils appeared first, followed by weakness of the limbs, but it took twenty-five minutes for the more serious symptoms of paralysis of the legs to be developed. He noticed, also, difficult breathing and frequent respiratory movements, which were afterwards succeeded by a slowing of the respiration and of the action of the heart; death followed, preceded by convulsions. With the larger doses, death took place within an hour; one animal died in eight minutes, two in eighteen minutes, three in twenty minutes, and four in fifty-eight minutes. The smallest dose named did not cause death until eight hours and forty-seven minutes after it was administered. The physiological action, according to Kobert and others, after the first local corrosion by the caustic action of the alkaloid, causes excitation of the motor centers in the spinal cord, which is followed very rapidly by paralysis. Coniin, like curare, first paralyzes the end of the motor nerves, afterwards their trunks, and, finally, the motor center. In the earlier stages, respiration is quickened, the pupils become contracted, and the blood pressure increases; but, on development of paralysis, the breathing becomes slower, the capillaries relax, and the blood pressure sinks. Death takes place, primarily from cessation of the respiration, the heart *ultimum moriens*. Coniin is eliminated by the urine, a part is decomposed in the body, and some passes off by the lungs.

700. Post-mortem appearances.—There are no characteristic signs, except the peculiar smell of coniin.

701. Tests and detection.—Coniin, like nicotin, is a colorless, volatile fluid, which, upon exposure to air and light, becomes darker and darker in color, and is partly transformed into a resinous substance. It has a very peculiar odor, said by many authors to resemble that of the urine of mice; this odor can be perceived by adding a little potassium hydrate to a few drops of the fluid extract of conium. Coniin is inflammable, and much less soluble in water than nicotin (about 100 parts of water); its aqueous solution has an alkaline reaction, and behaves in the same manner with the metallic salts as the aqueous solution of nicotin; if a saturated solution of coniin in water be warmed, it will become cloudy on account of the greater solubility of the alkaloid in cold than in warm water; it is also soluble in alcohol, ether, benzol, chloroform, amyl alcohol, and naphtha; unlike nic-

otin and the fixed alkaloids, it will coagulate albumin; it is also soluble in dilute acids, forming salts with them; the hydrochlorate may be obtained in the form of crystals, which show a beautiful play of colors when examined by polarized light. The most delicate precipitating reagents are solutions of potassio-bismuth iodid, of phosphomolybdic acid, and the solution of iodin in iodid of potassium, the last reagent precipitating coniin from solutions which contain only 1 part in 8,000 of water. (Dragendorff.)

Dr. Harley[62] relates a case in which it was impossible for him to distinguish, in urine from patients who had not taken conium, an ethereal extract treated with potassa, from an aqueous solution of conium which he used for comparison. He mentions this fallacy in order to show, "that, in examining the animal fluids or tissues for coniin, we must bear in mind that the addition of caustic potash to them will often develop an odor indistinguishable from coniin, and that nothing short of the isolation of the principle itself should satisfy us."

Coniin resembles, in many respects, bases which have been isolated from purefactive products, and great care must be exercised in differentiating this base from various ptomains and leukomains.

A glass rod moistened with hydrochloric acid, when placed near a solution of the alkaloid, produces a white cloud, and the coniin is converted into a crystalline mass of white needles. (Erhard.)

A drop of concentrated alcoholic solution of phenol phthalein added to the suspected liquid gives a red color if coniin is present, but no reaction with nicotin; the difference is more marked on adding chloroform.[63]

On passing dry chlorin gas over coniin, the latter acquires a purple-red to blue color. (Liebig.)

702. Isolation.—The isolation of coniin from organic mixtures is accomplished in precisely the same manner as that of nicotin, and the alkaloid remaining after the evaporation of the naphtha can be tested by the above tests.

Coniin is eliminated very rapidly by the kidneys, and can be detected in the urine during life. Dragendorff has succeeded in detecting it in the blood and in the liver also. It withstands decomposition of the tissues for a comparatively long period, and has been detected in the body of an animal several months after death. (Otto.)

The following process has been employed by Dr. Harley[64] for the

[62] The Old Vegetable Neurotics, the Gulstonian lectures of 1868. Macmillan & Co. London, 1869.

[63] Heut. Arch. d. Pharm. 1893, CCXXXI.

[64] Op. cit. p. 18.

recovery and detection of very small quantities of coniin when mixed with organic matters: "The substance known or suspected to contain coniin is comminuted and exhausted, after a few days' maceration in a percolator, by water acidulated with one fiftieth of its bulk of sulphuric acid; the filtrate is spread out in a thin layer upon flat dishes, and allowed to evaporate to the consistence of a thin syrup, in a warm, dry room, or at the distance of 3 or 4 feet before a fire; the residue is mixed with an equal bulk of a strong solution of caustic potassa (1 part to 3 of water), transferred to a long tube, and agitated with its bulk of ether several times during twenty-four hours. The ether is then decanted, and the alkaline mixture washed again and again with fresh portions of ether, until the coniin is completely removed. Two, or at most three, washings are sufficient for this purpose. On distillation of the ethereal solution, the coniin, more or less pure, remains. The impure coniin is next shaken with a small quantity of dilute sulphuric acid, which separates the alkaloid from oily or resinous impurities. From this solution of sulphate of coniin the base is separated in the usual way, *viz.*, evaporation to a syrupy consistence, mixture with caustic potassa, washing the mixture with ether, evaporation of the ether, and, finally, distillation of the coniin in a current of hydrogen, which may, of course, be omitted when we only want to determine the presence of coniin. If spirit be used in the exhausting process, the ethereal extract will be contaminated by much fatty and resinous matters; hence the advantage of using a watery solution. In searching for coniin in organic mixtures the same process may be adopted. The contents of the stomach may be digested for a few days with a sufficiently large quantity of sulphuric or oxalic acid to prevent decomposition, then strained, evaporated spontaneously, and treated as above. Before we conclude that coniin is present, we must isolate an oily matter which possesses, in addition to a coniin odor, an intensely sharp, biting taste, and which dissolves readily, with loss of odor, in a drop of dilute sulphuric acid. We must pour off this drop into a clean tube, and redevelop from it a strong coniin odor, by the addition of a little of a strong solution of caustic potassa. If we depend upon the odor alone we may fall into error."

V. Cicutoxin; Cicuta virosa; Cicuta maculata; Œnanthe crocata.

703. Cicuta virosa, in general.—Cicuta virosa (water hemlock) is

also one of the poisonous umbelliferous plants; the roots of this plant have been eaten in mistake for wild parsnip.

704. Symptoms of Cicuta virosa poisoning.—The symptoms of poisoning are somewhat similar to those of coniin, such as vomiting, muscular twitchings, feeling of numbness, muscular weakness, paralysis, labored breathing, convulsions, and unconsciousness.

705. Treatment of Cicuta virosa poisoning.—The treatment for these cases of poisoning is similar to that for the treatment of coniin poisoning.

706. Statistics of Cicuta virosa poisoning.—Mr. Blyth quotes the fact that Falk collected 31 cases of poisoning by this plant, of which 14 died.

707. Lethal dose.—The minimum fatal dose is unknown; death occurs within four hours, or sometimes later, after poisonous symptoms appear.

708. Cicuta maculata (cowbane), in general. — Cicuta maculata (cowbane), or poison hemlock, also contains coniin, according to Peterson and Haines. It grows wild in parts of the United States, and is sometimes cultivated in gardens. The leaves have been mistaken for parsley. Coniin exists in the plant to the amount of 1 per cent, in combination with organic acids. The plant has been the cause of the accidental poisoning of animals as well as man.

709. Statistics of Cicuta maculata poisoning.—There was a case reported by Medical Examiner Jack (Nos. 6 and 7, in 1891) in his returns to the secretary of state, Massachusetts, which relates that two sisters, aged seventy and seventy-two years respectively, gathered some roots of poison hemlock in mistake for roots of sarsaparilla; after eating portions of these roots they were very soon overcome by persistent and violent vomiting, collapse, coma, and tetanic convulsions, terminating in their death within a few hours of each other. The roots were recognized as belonging to the plant Cicuta maculata. There was no autopsy.

710. Symptoms of poisoning from these plants.—As the symptoms of poisoning, and the pathological appearances following poisoning, from the before-named class of hemlock shrubs, are similar in character, it may not be amiss to make the following extract from the summary presented by Witthaus and Becker in their recent work on toxicology (Vol. IV. p. 709): "The water hemlock, or cowbane, which grows abundantly in the north of Europe and less commonly in the south of England, has been frequently confounded with Conium, which it resembles in appearance, but from which it dif-

fers widely in its physiological action. Early writers refer frequently to poisonings occurring by the root having been mistaken for parsnips or celery root, or by the herb having been used in the kitchen in mistake for parsley. One of the earliest of toxicological monographs is that of Wepfer, relating to the poisoning of eight children by the root. Similar accidents are of occasional occurrence at the present time, although they are rarely reported in medical literature. In the United States, similar accidental poisonings by the closely related Cicuta maculata, which is widely known as water hemlock, spotted cowbane, beaver poison, or musquash root, are not of unusual occurrence.

"The symptoms of poisoning by Cicuta resemble those caused by Œnanthe crocata more closely than they do those of Conium poisoning; the onset is sometimes rapid, sometimes delayed. These are nausea, vomiting, colicky pains, staggering, palpitation, and loss of consciousness with, at first, ptosis, dropping of the lower jaw, and pale face and lips. Soon there are twitchings of the facial muscles; the patient becomes uneasy, the respiration more accelerated; the paleness gives place to cyanosis, and the patient is attacked with the most violent convulsions, at first tonic in character, becoming clonic, of long duration (one to five minutes), during which there is marked opisthotonos of the head, neck, and trunk, and the legs are drawn up; there is gnashing of the teeth, and the lips are covered with a reddish frothy fluid. During the spasms the feces are passed involuntarily, and the urine is expelled in a strong stream; and at its height, the respiration, and, apparently, the heart's action, also, are completely arrested. During the intervals, which are of about ten minutes' duration, the face is pale; the saliva flows from the corners of the mouth; the extremities are cold, though the rectal temperature is normal; the respiration is accelerated and stertorous; the heart's action is irregular, with pause after every second, third, or fourth beat; the radial pulse is imperceptible; the pupils are widely dilated; the corneal reflex is absent; and the patient is entirely unconscious. In fatal cases death occurs during a spasm, from asphyxia, or the severity of the spasms abates, and the patient remains unconscious, and expires in coma. In nonfatal cases recovery is rapid after cessation of the spasms."

711. **Treatment.**—Empty the stomach of its contents immediately.

712. **Post-mortem appearances.**—The stomach shows signs of injection of the mucosa, which may extend into the smaller intestines; the lungs show ecchymoses; and injection of blood vessels is usually seen as after death by asphyxia.

713. Œnanthe crocata (water dropwort), in general.—Œnanthe crocata (water dropwort) is another of the poisonous umbelliferous plants, and has been eaten by animals in the pasture; oxen have frequently been killed by eating it. Dr. Luff quotes Cameron[65] as reporting that 43 oxen, feeding on some of the Œnanthe crocata, were seized with frothing at the mouth, shivering, difficult respiration with spasm, and, finally, death, which occurred in all of the cases.

714. Symptoms of Œnanthe crocata poisoning.—Symptoms in man are also given by Dr. Luff; as, first, abdominal pain, vomiting, diarrhea; second, slow and feeble pulse, dilated pupils, collapse, and cyanosis; third, delirium, insensibility, and convulsions.

715. Treatment of Œnanthe crocata poisoning. — Treatment for cases of poisoning by this plant should be the same as for Conium.

716. Post-mortem appearances.—Although there is inflammation of the stomach and intestines, there are no characteristic post-mortem appearances.

VI. Curarin.

717. Occurrence and properties.—Curare is an arrow poison used extensively in South America. The active principle is curarin, besides various other alkaloids of lesser importance.

It forms prismatic crystals, which have a very bitter taste and are faintly alkaline. It is easily soluble in water and alcohol, slightly soluble in amyl alcohol and chloroform, insoluble in ether or benzol.

Curare is a substance used in South America and, perhaps, also in Borneo as an arrow poison, under the name of "woorali." Its composition as an arrow poison is a secret known to the natives that use it. Europeans who have attempted to obtain a knowledge of the materials from which it is made are convinced that it is a product of the decoction of vegetable poisons in combination with snake poison. It is supposed that some of the curare is derived from different species of strychnos, while some toxicologists believe that the active principles of curare may be methyl compounds similar to those which have been artificially made, as, for instance, methyl strychnin, methyl brucin, all of which have the same action as that of curare.

Curarin has been prepared from the original decoction, by a process suggested by Prior, in a crystalline form, as long ago as 1865.

718. Action.—It is chiefly interesting as a poison from its physiological action. When given to animals, it produces, in a very short

[65] London Lancet, 1873.

time, complete paralysis of voluntary motion and reflex excitation; the animal dies from asphyxia, while the heart continues to beat; by continued artificial respiration, under these circumstances, the pulsations of the heart may continue for a long time.

719. Tests.—With concentrated sulphuric acid, curarin yields a pale violet solution, which slowly turns red.

With sulphuric acid and a minute crystal of potassium bichromate, a blue color is produced similar to the reaction with strychnin, but differs from the latter in that the blue is more permanent, and is not followed by the changing to pink and yellow, but changes to a more or less permanent red (Dragendorff). With strychnin the red is more transitory.

720. Introduction by subcutaneous injection.—The only interest it may have in toxicology is from its possible introduction by subcutaneous injection, as its ingestion by the mouth is not likely to produce, nor has it ever produced, fatal action. Should it be a subject for medico-legal investigation, it is probable that its administration has been accomplished by subcutaneous injection, and it should be looked for at the seat of injury, in the liver, and in the kidneys.

VII. Gelsemin.

721. Occurrence and Properties.—Gelsemin is a crystalline alkaloid obtained from the yellow jasmine. In addition there have been extracted from this root, gelseminin, an amorphous alkaloid, and gelsemic acid.

Gelsemin is a white, amorphous, alkaline body. It also occurs as small, colorless crystals, which are easily soluble in alcohol, chloroform, and ether.[66] It forms crystalline salts with chlorin and bromin. This alkaloid is only very slightly toxic, inducing the same symptoms in frogs as strychnin, but having no action, even in large doses, upon mammals.

Gelseminin is a colorless, amorphous base, which turns yellow on contact with acids. Its salts are amorphous, and soluble in alcohol and water. It is a powerful poison which resembles coniin in most of its effects.

Gelsemic acid was first isolated by Wormley.[67] Robbins[68] and Schwartz[69] consider, however, that gelsemic acid is identical with the glucosid esculin, while Prof. Wormley, after a careful reinvestiga-

[66] Merck Bericht, 1891, ex Ph. Jhrber. 1891, p. 539.

[67] Am. Jour. Pharm. 1870.

[68] Ber. der Deutsch. Chem. Ges. 1876, 1182.

[69] Inaug. Dissert. Dorpat, 1882.

tion, disagrees with them,[69a] stating that it differs from esculin in regard to its crystallization, its solubility in water, ether, and hydrochloric acid, and its reactions with corrosive sublimate, sulphate of copper, a solution of bromin in hydrobromic acid, and in its physiological action.

Gelsemic acid is soluble in 2,912 parts of water at the ordinary temperature, in 330 parts of ether, and is rather freely soluble in chloroform. It is also soluble in the alkaline hydrates, the solution having a yellow color by transmitted light, but appearing greenish-blue by reflected light. Nitric acid dissolves it, giving a yellow-colored solution, which changes to deep red when treated with ammonia water. Sulphuric acid dissolves it, forming a yellowish solution, which does not change color on warming to 100°C.; from this solution, ammonia water, in excess, will produce a precipitate of white crystalline needles even with exceedingly small amounts (1/10000 gr.). Corrosive sublimate gives a copious yellow precipitate, from which large tufts of needles quickly separate. A solution of bromin in hydrobromic acid gives a green precipitate, which quickly changes to a blue, and finally to a brown. Sulphate of copper gives a dirty brown precipitate, which soon changes to a dull red color, and needle-shaped crystals separate. Acetate of lead gives a yellow precipitate, from which star-shaped groups of crystals separate; these crystals are insoluble in ammonia water, but soluble in acetic acid.

722. Preparations.—(U. S. P.) Rhizome and roots of Gelsemium sempervirens are used to make the following: Fluid extract of gelsemium; tincture of gelsemium containing an alkaloid gelsemin in proportion, estimated by Wormley, of 2 per cent. Thompson, of Detroit, discovered an allied alkaloid which he named "gelseminin." Prof. Wormley also separated a slightly acidulous constituent which he termed "gelsemic acid;" this has later been identified by Schmidt as beta-methyl esculin, which is found in other plants.

A "specific gelsemium," which is prepared from the green root by adding 10 gallons of alcohol to a barrel of cut roots, is used by eclectic medical practitioners and others.

723. Statistics of poisoning.—Prof. Wormley, in 1893, reports 25 cases of gelsemin poisoning, collected by himself, of which 13 proved fatal, the fatal period varying from one to eight hours.

There are five cases recorded[70] of fatal poisoning by the drug, and

[69a] Med. Jour. Pharm. July, 1882.

[70] Med. Rec. N. Y. 1882, 65; Boston 22, 1881; and Am. Jour. of Med. Sci. Apr. 1870. Med. and Surg. Jour. July 3, 1879, Dec.

several where serious, though not fatal, symptoms, followed various sized doses.[71] Its poisonous principle appears to reside in an alkaloid gelsemin, which, with gelsemic acid, has been chemically isolated by Prof. T. G. Wormley,[72] as above stated.

724. **Symptoms of poisoning.**—Five to fifteen drops of the fluid extract of gelsemium will cause muscular weakness and languor in most persons, though the susceptibility varies with the individual. After larger and poisonous doses, ½ to 1 teaspoonful of the fluid extract, there succeed to the above symptom certain nervous phenomena, such as dizziness, disturbed vision, and, sometimes, frontal headache; these symptoms may be intensified, amounting almost to muscular paralysis and blindness; the gait becomes tottering, the eyelids droop or cannot be opened, the eyeballs squint, and the muscular power of speech is lost; the cutaneous sensibility is diminished, respiratory movements and pulse become slower and weaker, a cold perspiration breaks out, and the bodily temperature is lowered. These symptoms may be observed twenty minutes after the taking of the drug, but in nonfatal cases they usually disappear in a very few hours. Convulsions are usually absent in man, though they have been observed in experiments on the lower animals. Death usually occurs from paralysis of, and absence of reflex action in, the respiratory nerve center. In one case reported by Dr. Boutelle[73] toxic symptoms appeared in half an hour, and the patient died in less than three hours. In the second, reported by Prof. Wormley, that of a pregnant woman, the symptoms did not appear for two hours, and she died in seven and a half hours.

725. **Lethal dose.**—Twelve minims (0.71 c.c.) of the fluid extract caused death in a child three years of age. Three teaspoonfuls of the fluid extract caused the death of a woman. Fifteen grains (.92 gm.) of a dried extract, known as "gelsemin resinoid," caused death in an adult. A healthy woman took an amount of concentrated tincture, which was equal to 1.6 grains (11 neg.) of gelsemin, and died in seven hours.

726. **Treatment.**—The stomach should be emptied and rapidly washed out with warm water; the circulation of the blood should be excited by internal and external stimulants, such as hot drinks, friction, and external heat; artificial respiration should be practised, and the patient should be urged to breathe. As the symptoms of

[71] Boston Med. and Surg. Jour. Feb. 9, 1871; and *Ibid.* Apr. 15, 1869.

[72] Am. Jour. Pharmacy, 1870.

[73] Boston Med. and Surg. Jour. Feb. 9, 1871; and *Ibid.* Apr. 15, 1869.

poisoning are transitory, in keeping up the circulation and respiratory movements by every means possible, the recovery may be promoted.

727. Action.—Gelsemin has a powerful influence in suspending the movements of respiration. Drs. Ringer and Murrell,[74] in experiments on cats, noted that in a few moments the respirations were slowed down to 12, and even 8 per minute. The lower half of the body suffered from loss of muscular power, while the upper half was convulsed during short intervals. In about half an hour the hind legs were almost completely paralyzed, and the respiratory movements were so shallow that they could hardly be counted. In the case of a dog, after all respiration had ceased, tracheotomy was performed and artificial respiration practised; the animal recovered. These experimenters concluded that gelsemin acted on the motor reflex system through Setschenow's inhibitory center, and destroyed the reflex power by its direct action on the motor nerve. Dr. Burdon-Sanderson also investigated the action of gelsemin on the respiration,—especially in reference to the movements of the diaphragm of rabbits. The animal was first narcotized with chloral, and a small spatula passed in through the peritoneal cavity by an opening along the linea alba, and upwards, in front of the liver, until its convex surface rested against the underside of the centrum tendineum. By attaching a lever to the free end of the spatula he could study the movements of the diaphragm, which were graphically recorded. The first influence seen from the action of the drug augmented the depth, but not the frequency, of the respiratory movements; the next, diminished the action of the diaphragm, both in extent and in frequency. As in other instances observed by physiologists, the overexcitation of a central organ resulted in paresis of that organ. In the case mentioned by Sanderson, the progressive diminution of movement was complicated with phenomena of asphyxia, which resulted in making the blood more and more venous. Dr. Sanderson concluded that the drug acted by paralyzing the automatic respiratory center, the gradual and progressive action of which was complicated by the imperfect arterialization of the circulating blood. Besides the above effects noted in animals, and the slowing of the respiratory movements, there is observed in man a particular action on the motor nerves of the eye, so that objects cannot be fixed, and they apparently dodge their positions; the eyelids become paralyzed; they droop, and cannot be raised by voluntary effort; the pupils are widely dilated, and

[74] London Lancet, Vol. I. 1876, p. 415.

there is a peculiar feeling of lightness about the tongue; it ascends, gradually, to the roof of the mouth, and speech is slurred. The effect on the lower half of the body noted in animals is also shown in man, the legs dropping down and refusing to support the body. Though the respiration becomes labored, as well as slowed, the pulse rises in frequency from 120 to 130 beats per minute, the mind remaining clear. The above symptoms appear in about an hour and a half after taking an overdose of the drug. If the dose is not fatal, the symptoms soon disappear without leaving any discomfort. If, however, the case becomes fatal, there may be convulsions, and death may occur very much as described in the experiments on animals. Large doses are very apt to produce a form of tetanic spasms, which are always preceded by loss of voluntary reflex power; the respiration ceases before the onset of convulsions, while the lower extremities are most affected, contrary to convulsions produced by strychnin; irritation fails to excite another paroxysm till a lapse of some seconds, just as if the exhausted nerve center required time to renew its energies; finally, the convulsions last only a short time.[75]

728. Tests and detection.—When a small portion of a residue containing gelsemium alkaloid is moistened with concentrated sulphuric acid, and a minute crystal of potassium bichromate is added, a red to purple color is formed in streaks along the path of the crystal.[76] "In a parallel experiment carried on with strychnin, the two alkaloids cannot be mistaken."[77]

A concentrated solution of cane sugar added to the residue, and then sulphuric acid added, develops a red color. Strychnin does not give this test.[78] With ferric chlorid and ferric cyanid, gelsemin gives an intense green color; strychnin does not react (Brouardel-Boutmy).

The physiological gelsemin alkaloids cause marked symptoms in 1/8 grain (0.008 gm.) doses administered hypodermically to a cat.[79]

729. Isolation.—Gelsemin, gelseminin, and gelsemic acid can be isolated from organic mixtures in the same manner as mentioned in speaking of opium (see *infra*, § 773).

The isolation of the above principles from organic mixtures can be accomplished in the same manner as mentioned in speaking of opium.[80] The gelsemic acid can be removed from the acid aqueous fluid by shaking it either with ether or chloroform, which solvents,

[75] Blyth, op. cit. p. 347.
[76] Wormley, Am. Jour. Pharm. 1882, IV. 721.
[77] Gerrard, *Ibid.* 1883, 258.
[78] Witthaus and Becker, Toxicology, 342.
[79] Wormley, loc. cit.
[80] See § 773.

upon evaporation, will leave it in the form of a crystalline residue. The gelsemin can be separated from the alkaline, but not from the acid aqueous, solution by shaking with ether or chloroform, which, after separation from the aqueous fluid and evaporation, will leave it as a residue. These residues can be tested by the above tests.

VIII. Nicotin.

730. Occurrence.—Although there are three distinct forms of the Nicotiana, the Havana, French, Dutch, and American tobaccos are mostly from the Nicotiana Tabacum. This plant contains a volatile, poisonous liquid alkaloid called "nicotin." This alkaloid is apparently united with citric and malic acids in the plant. According to chemists, the amount of nicotin contained in commercial tobaccos varies from 1.5 per cent to 7.96 per cent. The Havana and Maryland tobaccos contain about 2 per cent, the French tobacco varies from 3.22 per cent to 7.96 per cent, the Paraguay contains from 1.8 per cent to 6 per cent, and the Algerian contains probably a little less than 9 per cent.

731. Accidents, and poisoning from use of.—Mr. Blyth reports 4 cases of accidental fatal poisoning and 1 case of suicide from tobacco.

Many accidents have occurred from the swallowing of crude tobacco, and from children playing with old pipes. Many of these cases resulted fatally. Tobacco juice is not nicotin, but it probably contains a very large amount of it.

732. Symptoms of poisoning, in general.—The symptoms reported by Dworzak and Heinrich were those which follow the first dose of 1 milligram; *viz.*, an unpleasant sensation in the mouth and throat, salivation, and an uncomfortable feeling spreading from the epigastrium to the extremities of the body. After a dose of 2 milligrams there were headache, giddiness, numbness, amblyopia, torpor, the sensation of hearing dulled, and rapid respiratory movements. After doses of 3 to 4 milligrams there appeared in forty minutes a feeling of great faintness, physical depression, and weakness; the limbs were cold and the face was pale; this was followed by nausea and diarrhea. In one subject one of the two experimenters noted a shivering of the extremities, and cramps in the muscles of the back, in addition to difficult breathing; another subject suffered from muscular weakness, felt faint, had fits of shivering, and creeping sensations about the arms. Though the severer symptoms gradually abated in two or

three hours, recovery was not complete for two or three days. It has been observed that, when death occurs, the heart continues to act though respiration has ceased. Nicotin has had much interest attached to it from having been the poison used by the Count of Bocarmé in the murder of his brother-in-law, Gustave Fougnies. The nicotin was obtained by Professor Stas from the mouth and stomach of the deceased, and from articles of clothing and furniture. It had been prepared by the hands of the murderer himself, who had devoted several months to the study of the process of extracting it from tobacco. The symptoms produced by it in the human system were then not well known. Besides the one already referred to, there is but a single case on record, and in that, also, the symptoms were not witnessed. From the circumstances it was inferred that the person suddenly became insensible and powerless, and died in from three to five minutes.[81] In experiments upon dogs, Orfila observed that vertigo was first produced, that they then sank down, had tetanic convulsions with opisthotonos, and died in a variable time, according to the strength of the liquid. Anhydrous and pure nicotin, he says, may kill a dog in half a minute, but two minutes are usually required. If somewhat less pure and more diluted, death will follow in about ten minutes; and, if still further diluted, the animal may recover. The doses given were from 1 to 12 drops.

733. **Symptoms of acute poisoning.**—The symptoms produced by a poisonous dose of tobacco are nausea, vomiting, a burning heat in the throat and stomach, colic, diarrhea, frequent urination, extreme giddiness, great anxiety with a disposition to faintness, pallor, coldness of the extremities, and spasmodic trembling; the pulse is small, weak, tremulous, and intermittent; the breathing is labored and stertorous; there is a paralytic relaxation of the voluntary muscles, with clonic spasms of the limbs. The pupils are but slightly affected, and the eyes seem to be sensible to light. This state is succeeded by a general torpor, or utter prostration, which is not coma, but which may terminate in death. Tobacco has produced death by having been criminally mixed with liquor. The dose capable of destroying life, when introduced by the mouth, is not known.

Two cases are related by Dr. Deutsch, in which life was in extreme danger from the swallowing of tobacco. In one, a soldier, suffering with a tapeworm, took, by the advice of a friend, some of the extract of tobacco, such as is deposited in smoking pipes. The

[81] Times and Gaz. June, 1858. p. 659;
Guy's Hospital Reports, 3d ser. IV. 345.

quantity swallowed was estimated to be 1 ounce (31 gm.). He was at once seized with the most horrible pains in the stomach, and fell into a state of extreme collapse. The efforts to vomit were ineffectual until an emetic was given to him. After extreme suffering he slowly recovered. In the other case, a young lady accidentally swallowed the lighted stump of a cigar which she had been smoking; she suffered greatly from the ordinary symptoms of poisoning by tobacco together with pain in the stomach, until she was relieved of it by vomiting. A fatal case of a man who swallowed a large mouthful of crude tobacco is reported by Mr. Skae. In addition to the usual symptoms he had convulsions.[82] A further variation from the ordinary course of symptoms was shown in the case of two females, about eighteen years of age, both of whom drank an equal quantity of a decoction of tobacco. The one was affected in the usual manner. The other became insensible and was attacked with convulsions; her arteries and veins were distended, the former throbbed forcibly, and the conjunctiva was injected.[83]

Dr. Weaks, of Vermont, mentions the case of a child, a few days old, to whom two tablespoonfuls of water impregnated with the smoke of tobacco were given for the purpose of keeping it quiet. It died comatose in eight hours, notwithstanding the most active efforts for its resuscitation.[84]

Pereira quotes from Dr. Copland an instance in which ½ dram (2 c.c.) administered by enema proved fatal, and other cases in which 1 and 2 drams (4 and 8 c.c.) had the same effect. In one of these referred to by Dr. Christison, death occurred in thirty-five minutes. Dr. Travignot witnessed a fatal result in the case of a robust man, fifty-five years of age, who took an enema prepared from 15 grains (1 gm.) of tobacco.[85] Dr. Eberle saw the life of a boy destroyed in less than twenty minutes by a tobacco enema.[86] Tobacco can also be absorbed when used as a poultice upon the skin. Several instances are recorded, in which the external application of moistened tobacco leaves produced alarming symptoms or death (*vide ante,* Chap. I, § 8). Two deaths are said by Gmelin to have resulted from excessive smoking, in one case seventeen, in the other eighteen, pipes having been smoked at a sitting. The smoking of tobacco is followed by severe symptoms in some persons who have an idiosyncrasy which makes them more susceptible than others to the action of tobacco.

[82] Edin. Med. Jour. I. 643.
[83] Dierbach, Neueste Entdeck. II. 884.
[84] Boston Med. and Surg. Jour. Vol. XLVII. p. 461.
[85] Rev. Méd. Nov. 1840.
[86] Therapeutics, p. 389.

Blyth also quotes the two cases of Gmelin, in which the death of two brothers followed the smoking of seventeen and eighteen pipefuls, respectively, of German tobacco.

Vohl and Eulenberg made some very careful experiments on the character of the smoke coming from strong tobacco, whether burned in pipes or in cigars.[87] After passing the smoke through potassa, which absorbed all the volatile substances in the smoke, the vapor was then passed through diluted sulphuric acid, which held the base of a substance called "pyridin;" this is also an alkaloid obtained from the cuttle fish by Gautier's process.[88] The action of this bases, as investigated by several observers, has been discussed in the 4th chapter, under the head of "Pyridin." As the peculiar action of tobacco is represented in cases where nicotin has been used, it would be better to describe the characteristics of the latter as representing poisoning from tobacco. Poisonous action follows the absorption of pure nicotin, with the same rapidity as prussic acid; but, of course, where occurring in crude tobacco, its absorption is more slowly accomplished, and, though poisonous, the rapid action is not so remarkable. Enemata of tobacco have caused death in fifteen and eighteen minutes; with the pure alkaloid nicotin taken by the mouth, in five minutes. Dworzak and Heinrich both made experiments upon themselves with pure nicotin.

The subacute and chronic symptoms of tobacco poisoning hardly belong to a treatise on toxicology. Kobert gives, however, as symptoms of chronic poisoning from tobacco, catarrh of the pharynx, larynx, and middle ear, violent beating of the heart, limitation of field of vision, scotoma, weakness of memory, dizziness, gastralgia, and sclerosis of the arteries. It is fair to say that many people addicted to the use of tobacco do not generally suffer from the symptoms given by Kobert.

734. Lethal dose.—This is given by Kobert as 1 grain (0.06 gm.) of nicotin, and as 13 grains (0.8 gm.) of snuff administered internally. The poisonous dose of tobacco leaves seems to be ½ to 1 ounce (15 to 31 gm.).[89]

735. Treatment.—In the case of nicotin there would hardly seem to be time for any remedial action, as the absorption of nicotin into the circulation is concentrated and rapid. In the case of tobacco being swallowed, the first indication would be the washing out of the

[87] Arch. Pharm. CXLVI. p. 130.

[88] Comptes Rend. CVI. 852, 861; CVIII. 58, 59; 809, 810; CVI. 1604, 1605.

[89] Edin. Med. Jour. 1855.

stomach with cold water, and, possibly, the administration of powdered charcoal to collect the finely divided particles of tobacco; if snuff is swallowed, the stomach should immediately be washed out again. Stimulants, external warmth, artificial respiration, or the inhalation of oxygen gas; and sulphuric ether, brandy, tannin, or an infusion of tea may be administered; possibly, as a heart stimulant, it might be advisable to use, by subcutaneous injection, a very minute dose of strychnin, or, by the mouth, tincture of nux vomica in doses of 30 drops.

736. **Action.**—According to Dragendorff, nicotin is absorbed into the blood and eliminated as such unchanged in part by the kidneys, and in part by the saliva. Recent observations by Schweinitz and Edsall[90] would tend to show that action upon the ganglionic cells of the retina and the optic nerve is produced by liberating a toxic substance in the system by some method of auto-intoxication, just as in the case of anilin, wood alcohol, and picrotoxin. These writers quote the opinion of Horner, that neither alcohol nor tobacco, as such, is a direct toxic agent in cases of central amblyopia; and also that of Sachs, who maintains that, even in pure tobacco poisoning, some complex chemical decompositions occur in the stomach, and that from these there is a resulting transformation of the gastric juices, which, combined with nicotin, form substances which are more poisonous than the simple tobacco bases themselves. Whether this theory, as presented, is true or not, it does not seem to appear that this particular class of protoplasmic poisons acts directly as such, but that the action is due to some secondary auto-infection, which, at present, is mysterious; for, after an intermission of the use of tobacco when these effects on the retina have been observed, the disturbance of vision does not immediately clear up, but requires some special medicinal treatment for its cure. It has been noticed in some of these cases that, coincident with the condition of amblyopia, the biliary matters passed out with the urine, yet in none of these cases was there any glycosuria. The intimate action, therefore, upon the chemico-physiological processes concerned in the metabolism of the tissues is, at present, in an unknown condition. We note from the researches of Rosenthal and Krocker[91] that nicotin, whether by the simple or the complex process above described, acts energetically on the brain, at first by increasing its excitability, and afterwards, ac-

[90] Transactions of the Am. Ophthal. Soc. Vol. X. Pt. 1, 1903.

[91] Weber, Die Wirkung des Nikotines auf den Thierischen Organismus, Berlin, 1868.

cording to the rule in such effects, from exhaustion, resulting in lessening its activity. The same action is exerted upon the spinal cord. The resulting convulsions appear to have a central origin in the brain. Muscular irritability is not destroyed, and the heart continues to beat—especially the auricles—after cessation of respiration. Artificial respiration does not seem to influence the convulsions. The section of the vagus nerve has no influence in checking the peculiar respiratory movements, which at first are quickened, and then slowed, and, lastly, they cease. The cause of death, apparently, is from asphyxia, due to paralysis of the respiratory center. It should be particularly mentioned that death does not follow on account of the failure of the heart's action, although nicotin does powerfully affect the heart's action; small doses of nicotin seem to excite the termination of the vagus nerve in the heart, and cause a slowing of the beats. Large doses, apparently, paralyze both the controlling and exciting nerve centers, whose influence upon the arterial blood vessels produces a reduction of their caliber, and, afterwards, a dilatation of these capillaries; as a consequence of this, the blood pressure will at first rise and then fall. Nicotin has a peculiar action upon the involuntary muscles of the intestines,—especially of the smaller intestine, the caliber of which is reduced throughout its whole length. This is ascribed to the excitability of the ganglionic cells and nerves of the intestines. The uterus, also, is excited to a strong contraction by nicotin. The salivary and biliary secretions are increased.

737. **Post-mortem appearances.**—Except for the contraction of the lumen of the intestinal canal, there seem to be no especial appearances to mark distinctively nicotin or tobacco poisoning. The detection of poisoning from nicotin will depend upon the chemical investigation.

738. **Physiological tests.**—For the physiological tests, the frog is the most susceptible to the action of nicotin, which is very characteristic.

739. **Isolation.**—Nicotin can be isolated from organic mixtures by the same process recommended in the case of opium (see § 773), naphtha removing it from the alkaline solution.

Although nicotin is volatile, it does not readily escape from the body after death, and it can be detected for some time. Melsens[92] states that he has been able to detect it in the body of an animal seven years after death. In cases of tobacco poisoning, the nicotin

[92] Gaz. Hebdom. de Méd. I. 1859.

is most readily detected in the contents of the stomach and intestines; but it can also be detected in the solid viscera, as the liver, heart, lungs, and kidneys. Dragendorff and Taylor have both succeeded in isolating it from the blood.

740. Properties and tests.—Nicotin is an oily, transparent, colorless liquid, becoming brown and thick upon exposure to the air, and, when pure, exhaling a slight smell of tobacco. Its taste is acrid and burning. The vapors that are given off, when it is volatilized at 200°, have so strong an odor of tobacco, and are so irritating, that, according to Orfila, it is difficult to bear them. Nicotin is soluble in alcohol, water, ether, and the oils. Its aqueous solution has a strong alkaline reaction, and behaves in many respects like ammonia water when added to solutions of metallic salts, as, for instance, when added in excess to a solution of sulphate of copper, it produces an azure-blue color. It is soluble in any proportion in water, and can be partly precipitated from a concentrated solution by potassium hydrate. It is also quite soluble in dilute acids, forming salts with the acids. Nicotin exposed to the air and light soon turns yellow and then brown, a resinous substance being formed. It is readily precipitated from its acid solutions by phospho-molybdic acid, and from its neutral solutions by Mayer's reagent, [93] trichlorid of gold, bichlorid of platinum, tannic acid, corrosive sublimate, and a solution of iodin in iodid of potassium; if an ethereal solution of nicotin be added to an ethereal solution of iodin, an amorphous brown precipitate is first formed, which, in a few hours, becomes changed into a crystalline one, the crystals sometimes being ½ inch in length. (Roussin's test.)

On heating gently with a little HCl, a violet color is developed, which changes to orange-red on adding HNO_3. (Palm.)

Add a few drops of 30 per cent formaldehyd, free from formic acid, to nonresinified nicotin, then add a drop of HNO_3, the solution acquires an intense pink color. If much nicotin is present, it becomes a dark red color; if nicotin is resinified, the color is blood red. (Schindelmeiser.) The odor is one of the most characteristic of its tests.

If a glass rod moistened with hydrochloric acid be held over a watch glass containing the alkaloid, a white cloud is produced like that with coniin; but no crystals are produced as with the latter.

Gold chlorid produces an amorphous precipitate in neutral solutions of nicotin salts, which is nearly insoluble in acetic or hydro-

[93] See § 757.

chloric acid, but readily soluble in caustic alkalies. A cloudiness is produced in a solution of one part in 10,000.

IX. Strychnin and brucin.

741. **Occurrence.**—Strychnin is the principal alkaloid occurring in several species of Strychnos, the most common of which are Strychnos Nux vomica and Strychnos Ignatii. It is found in largest amounts in the seeds, but also occurs in small quantities in the bark. Strychnin is always accompanied by another alkaloid, brucin, which produces milder but similar symptoms to those of strychnin.

742. **Properties and tests in general.**—Strychnin occurs mostly in the form of prismatic crystals belonging to the rhombic system, when allowed to crystallize slowly; but when it separates quickly from concentrated solutions, the crystals are needle-shaped. It is soluble in about 7,000 parts of cold, and 2,500 parts of boiling, water, but is readily soluble in alcohol, amyl alcohol, benzol, naphtha, and chloroform; the last solvent dissolves it the most readily, taking up nearly 20 per cent of its weight; it is but slightly soluble in ether; it is readily soluble in dilute acids, forming salts which are readily soluble in both water and alcohol. If strychnin is gently heated, it volatilizes unchanged, and can be condensed in the form of a sublimate upon a glass microscope slide or upon the inner surface of a glass tube; this sublimate assumes the characteristic crystalline form if touched with a drop of water. All solutions of strychnin have an exceedingly bitter taste, which can be perceived if a single drop of a solution containing only 1 part of the alkaloid in 100,000 parts of water be introduced into the mouth, and, according to some authors, it can be perceived when the solution contains only 1 part in 420,000. It is the most bitter substance known, and it is very difficult to disguise the bitter taste; so that it can only be administered criminally by making the victim believe that he is taking some very bitter medicine. The writer is familiar, personally, with two instances in which an attempt was made to poison several persons by introducing strychnin into tea; but it was unsuccessful, since the introduction of the first teaspoonful into the mouth showed, by the intense bitterness, the presence of some unusual substance, and, if swallowed, the amount taken was only a small medicinal dose. Strychnin is readily precipitated from its solutions in acids by ammonia water, usually in crystalline form; this test (and the same is true of the other tests which produce precipitates) is best performed

by placing a drop of the solution upon a glass slide, adding a drop of the reagent and stirring the mixture with a glass rod; the crystals formed can then be examined with a microscope. Strychnin treated with a drop of concentrated sulphuric acid forms a perfectly colorless solution, which, if stirred with a crystal of potassium bichromate, passes through a beautiful play of colors; first a purple color is formed immediately; this changes quickly to a violet, then to a red, then to a yellow, and finally the mixture becomes colorless, unless too much of the bichromate has been added. This test gives about the same play of colors with curarin, but the red is produced much more quickly, and is much more permanent than in the case of strychnin; moreover, in legal cases there is no danger of confounding these two substances, since they are separated from their solutions at different stages of the process for the isolation of organic poisons (see § 773).

In place of potassium bichromate, other oxidizing agents, such as cerosoceric oxid, potassium permanganate, manganese dioxid, and lead peroxid, may be used. The writer has sometimes noticed that, when he was testing for strychnin in a given residue, he obtained a faint blue color on the addition of a crystal of potassium bichromate, which did not fade, but persisted for several days, occasionally becoming deeper. This was more often the case, if, in the extraction of the original material, oxalic acid had been used instead of sulphuric acid. When the residue was heated with a drop or two of sulphuric acid, again extracted if necessary on account of the charring, then cooled, and the bichromate was applied, the characteristic play of colors was obtained, if strychnin was present.

Prescott[94] states that a distinctive test can be obtained with 0.0000025 gram strychnin; Wormley gives the limit as 0.000007 gram; and Hinsdale, with cerosoceric oxid, as 0.0000007 gram.

Strychnin is precipitated by a solution of potassium bichromate into the form of golden-yellow groups of crystals made up of an aggregation of needle-shaped crystals; this precipitate can be obtained with $\frac{2}{100}$ of a milligram (less than $\frac{1}{3000}$ of a grain).

A solution of iodin in iodid of potassium gives a brown amorphous precipitate, which is soluble in boiling alcohol, and separates from it upon cooling into the form of prismatic crystals; this test is delicate for $\frac{12}{10000}$ of a milligram (less than $\frac{1}{50000}$ of a grain).

Picric acid with a solution of strychnin gives a golden-yellow precipitate, which is amorphous at first, but gradually becomes crystal-

[94] Peterson and Haines, loc. cit. p. 518.

line, the crystals having an irregular but peculiar shape; this test is delicate for $\frac{5}{100}$ of a milligram (less than $\frac{1}{1000}$ of a grain).

Ferricyanid of potassium with a solution of strychnin gives an amorphous precipitate, which quickly becomes converted into groups of greenish crystals having a very peculiar form.

Corrosive sublimate also produces a precipitate, which is amorphous at first, but which soon becomes converted into groups of colorless, prismatic crystals.

Mayer's reagent gives a precipitate which is amorphous at first, and then becomes crystalline.

Trichlorid of gold and bichlorid of platinum also yield crystalline precipitates with a solution of strychnin.

The valuable negative tests are that neither Fröhde's reagent nor concentrated nitric acid gives a colored solution with pure strychnin.

743. Properties and tests of brucin.—Brucin crystallizes into the form of oblique rhombic prisms, which are more soluble in water and alcohol than strychnin; it is insoluble in ether, but soluble in benzol, chloroform, naphtha, amyl alcohol, and the dilute acids; alkalies precipitate brucin from its solutions in acids, usually in the form of an amorphous precipitate, which gradually becomes crystalline; it is quite soluble, however, in an excess of ammonia water. Concentrated nitric acid, or concentrated sulphuric acid, which contains a trace of nitric acid, imparts to brucin a beautiful orange-red color, which is changed by a solution of stannous chlorid to a reddish-violet; this test is delicate for $\frac{1}{100}$ of a milligram (less than $\frac{1}{6000}$ of a grain). It is precipitated into an amorphous form, the precipitate gradually becoming crystalline by Mayer's reagent, potassio-cadmic iodid, bichlorid of platinum, picric acid, and potassium bichromate. Pure concentrated sulphuric acid and Fröhde's reagent impart to it a pale rose color. It has a very bitter taste, but not as bitter as strychnin, and it is not nearly as active physiologically as strychnin. According to Sonnenschein,[95] it can be converted into strychnin by the action of oxidizing agents such as nitric acid; and, *vice versa,* strychnin can be converted into brucin by the action of reducing agents such as sulphid of ammonium.

744. Detection and isolation of these alkaloids.—The isolation of these alkaloids from organic mixtures can be accomplished by the same process as recommended in the case of morphin (see § 773), both the strychnin and the brucin being separated from the alkaline

[95] Ber. der Deutsch. Chem. Ges. 1875, No. 4.

solution by shaking with naphtha, benzol, or chloroform. Benzol removes them better than naphtha, and, upon evaporation, leaves the brucin as an amorphous residue near the edge of the watch glass, and the strychnin near the centre as a crystalline residue. Chloroform removes them better than either naphtha or benzol, and, upon evaporation, leaves both in the form of a crystalline residue. These residues can be tested by the above tests, the precipitating reagents being applied to a solution of the residue in water to which a little acetic or sulphuric acid has been added.

If both strychnin and brucin are detected, the inference is that the form of poison used was nux vomica or one of the pharmaceutical preparations of nux vomica; while, if either alkaloid alone is present, the source of poisoning must have been that alkaloid or one of its salts. Strychnin withstands the decomposition of animal tissues for a very long period, and has been detected in the body more than a year after death.[96]

The physiological test should never be omitted in cases of strychnin poisoning which are investigated legally; an exceedingly small amount of strychnin given hypodermically to a frog will produce the tetanic spasms; care should be taken not to give too large an amount, lest immediate death be produced; $\frac{1}{5000}$ part of a grain will usually suffice to produce the effect in a frog after a few minutes.

M. Prévost[97] contrasts the effects of veratrin upon the muscular system of frogs, in a way which may serve to test the presence of this poison as distinct from that of strychnin.[98]

The differential effects are as follows:—

In strychnin poisoning.	In veratrin poisoning.
1. Convulsions occur at the commencement.	1. Spasmodic contractions occur at the commencement.
2. The initial convulsion is succeeded by a series of convulsions.	2. The initial convulsion is prolonged, and usually terminates in a series of faint fibrillary twitches.
3. Extremely slight peripheral excitation causes a convulsion.	3. It is difficult to originate spasmodic contractions by excitation of the periphery.
4. The most feeble peripheral excitation always originates general convulsions.	4. Excitation often causes contractions that are altogether limited to the excited portions; occasionally, however, the contractions are general.
5. Convulsions disappear on destruction of the spinal cord.	5. Spasmodic contractions may be caused, even after destruction of the spinal cord, by irritation of either the nerves or the muscles.

[96] See Magoon case. Appendix, p. 788.

[97] Recherche Expérimentale relative a l'Action de la Veratrine.

[98] It may be well to state here that many of the alkaloids can be recognized by their effects upon the smaller animals.

6. Convulsions cease in limbs separated from the trunk and, consequently, from the spinal cord. In parts so separated, excitation of nerve trunks or of muscles causes merely normal contractions.

7. Convulsions occur in limbs separated from the circulation by ligature of the blood vessels, provided the nerve trunks are intact.

6. In limbs separated from the trunk and, consequently, from the spinal cord, spasmodic contractions may be caused by irritation of the nerve trunks or of the muscles.

7. No spasmodic contractions occur in limbs separated from the circulation by ligature of the blood vessels.

745. Statistics of poisoning.—Mr. Blyth ranks nux vomica and strychnin as the fifth on his list of substances which destroy life either by murder, suicide, or accident. His total for the ten years ending in 1892 is given as 201 cases of fatal poisoning from strychnin and nux vomica; of this number 8 were by murder, 150 were by suicide, and 43 by accident or negligence.

In the returns of the Massachusetts medical examiners for the 26 years ending in 1902, there are 48 deaths reported from poisoning by strychnin; of this number 2 were by murder, 27 suicidal, and 19 accidental or by negligence. Compared with arsenic, the number of deaths from strychnin and nux vomica is nearly double, according to Mr. Blyth's mortality record. The table of statistics in Massachusetts shows that the number of cases in which arsenic is used as a poison in the three classes with fatal results is nearly nine times greater than that of nux vomica and strychnin.

The extremely bitter taste of strychnin, even when sparingly soluble in water, is so very intense that it would be supposed that this poison could not generally be given to persons with intent to murder; but, out of 130 cases of poisoning during the last 25 years, collected from medical literature by Schauenstein, 15 were homicidal. It should be remembered that bitter ale will disguise the bitter taste of strychnin; and, as the solution of strychnin has no irritating property, a sufficient amount may be swallowed by a patient in one gulp to cause death; this is especially true when we consider that the bitter taste of strychnin is more of an after taste. There are four cases on record in England where Thomas Neill (his real name is Thomas Neill Cream), accused of having murdered four people between 1890–93, was actually convicted of causing the death of one, Matilda Clover, on conclusion of his trial, October 21, 1892. She was murdered October 20, 1891.

746. Preparations.—(U. S. P.) The seeds of nux vomica are required to make both the fluid and solid extracts of nux vomica. "These seeds are about 25 milimeters in diameter, orbicular, grayish or greenish-gray, soft, hairy, of a silk luster, with a slight ridge extending from the center of one side to the edge; internally horny.

somewhat translucent, very tough, with a large circular cavity into which the heart-shape nerved cotyledons project; inodorous and persistently bitter.

"The powdered seeds look not unlike licorice powder and, when met in its pure state, gives a dark orange-red color with nitric acid; this color is destroyed by chlorid of tin. The watery infusion gives a precipitate with tincture of gall, is reddened with nitric acid, and gives an olive-green tint with persulphate of iron. To recognize quickly nux vomica powder, if under examination, is to extract its strychnin by the following simple process: The powder is completely exhausted by boiling alcohol (90 per cent); the alcoholic extract is then evaporated to dryness and treated with water; this watery solution is then passed through a wet filter and concentrated by evaporation to a small bulk. To this liquid a drop or two of concentrated solution of picric acid is added; it will precipitate the yellow picrates; these treated with nitric acid, and the picric acid removed by ether, will, by the addition of soda, precipitate pure alkaloid, which can be shaken out by chloroform." (U. S. P.)

Solid extract of nux vomica, according to the United States Pharmacopœia, which gives an assay process, should be found to contain 15 per cent of the total alkaloid.

The fluid extract of nux vomica required by the United States Pharmacopœia should contain a little less than 24 grains (1.6 gm.) of total alkaloid.

According to the United States Pharmacopœia, 100 cubic centimeters of the tincture of nux vomica evaporated to dryness, and the residue tested by the same method of assay, should be found to contain 4.0 grains (0.26 gm.) of alkaloids.

Strychnin and sulphate of strychnin are the two preparations of strychnin required by the United States Pharmacopœia. The former is used to make two other preparations; *viz.*, citrate of iron and strychnin, and syrup of iron, quinin, and strychnin phosphates. Citrate of iron and strychnin is quite soluble in water, while strychnin itself is very sparingly soluble in water. The medicinal dose of the latter is from 1 to 3 grains (0.065 to 0.20 gm.) used cautiously. The medicinal dose of the syrup of phosphates of strychnin, iron, and quinin, is from 1 to 2 fluidrams (4 to 8 c. c.). Five grams, or a small teaspoonful, contain 1/30 of a grain (2 mg.) of strychnin, and about 1 grain (66 mg.) each of both quinin and iron.

747. Symptoms of poisoning.—Strychnin is the most violent in its action of all of the convulsive poisons, and, by comparison of its

least poisonous doses with those of brucin, is more than thirty-eight times as energetic, and kills an animal in less than one third of the period required for the latter alkaloid; as compared with the convulsive properties of the alkaloids of opium, it is twenty-four times more active than thebain, fifty times more active than laudanin, eighty-five times more than codein, and four hundred times more energetic than hydrocotarnin.[99] This less energetic action of brucin is in part explained (Binz) by its less poisonous effect, and, in part, because it is more rapidly eliminated by the kidneys. In a medicinal dose of 1/64 to 1/12 of a grain (0.001 gm. to 0.005 gm.), strychnin produces in man only the effects of a bitter stomachic tonic, increasing the flow of urine and of saliva; in larger doses, its poisonous action will appear, and has the same characteristics in all the vertebrate animals. This becomes an important fact in medico-legal investigations, since the recovery of the alkaloid from the body of a person, whose death has been caused by this poison, may often be in sufficient amount for use in causing the death of a small animal (a frog, for instance), and thus a physiological test may be added to the chemical proof. The phenomena of its toxical action will depend upon the quantity of poison swallowed and the state of fullness or emptiness of the stomach, since the fasting stomach will absorb more rapidly and larger amounts of the poison; occasionally an instance may occur, in which an individual may resist a dose which may be poisonous to the generality of persons. The first evidence of the absorption of a dose dangerous to life is an increased activity of all the senses, such as restlessness of mind and muscles, increasing agitation, sensations of itching, exaggerated sensibility to touch, the eyes showing an intolerance or increased sensibility to light, an increased sensibility to odors and taste. Soon after these early signs of nervous exaltation, muscular stiffness accompanied by twitchings will appear, with unsteadiness in walking; the muscles of the jaw are usually the last to be attacked with this stiffness or rigidity, which is often painful, and the temporal muscles, as well as those of the neck, are the seat of little convulsive movements. If the dose be large enough, these symptoms, which usually come on within the first half hour, are followed by more serious as well as severe muscular contractions of the whole body, which will be stretched out stiffly with the back arched, and supported

[99] Falck, Vierteljahr, f. Gericht. Med. 1874, p. 195; and Ann. d'Hygiène pub. et de Méd. Lég. 1876.

sometimes only by the head and heels (opisthotonos); frothy mucus collects about the mouth, the face is pale, articulation interrupted and difficult, the jaws locked, with the tongue sometimes between the teeth, while intellectual activity is perfectly intact; the limbs become agitated with violent and noisy contractions, which in criminal cases is often compared to the clapping of castanets (Tardieu, case of Pégard). These various phenomena of convulsions, after a longer or shorter continuance, culminate with the characteristic tetanic spasms; the muscles become rigid, and violently throw the limbs into forcible contraction, the sole of the foot is turned inwards, and the muscles of the face are distorted in horrible and anxious grimaces. The victim will complain of these painful contractions, and make vain efforts to raise himself, make inarticulate efforts to cry out, and experience a most lively thirst. In men there are painful erections of the penis, and in women corresponding sexual sensations.[100] Respiration is difficult, from the muscular rigidity of the thoracic muscles, the pupils are dilated, the neck is swollen, the lips are blue and cyanotic from the tendency to asphyxia, and, at the moment of impending death, respiration slowly reappears, the lips open, the victim apparently revives, and, if the dose be large enough, the sequence of symptoms will be repeated; but, if recovery is to take place, the spasms return with diminished force, and all convulsions will ordinarily cease in a few hours, leaving an intense muscular fatigue, a moral and physical languor, with increased excitability to reflex irritations, and often with a muscular stiffness which may persist for a longer or shorter time. Death takes place from asphyxia, caused by the tonic contraction of the thoracic muscles, in the most violent paroxysmal convulsions, or from exhaustion during the interval after the fourth or fifth paroxysm. The paroxysms usually last from one to five minutes, and the intervals from five to fifteen minutes; in cases which terminate in recovery, the paroxysms become less and less severe, and the intervals longer and longer, while the reverse is true in those cases which end fatally. Death usually takes place in adult human beings during or after the fourth or fifth paroxysm, but children may die during or after the first, second, or third convulsion. This is an important point in medico-legal cases in distinguishing between the convulsions of strychnin poisoning and uremic or puerperal convulsions (see §§ 749, 754). During the intervals, except in rare cases during the fourth, the patient is able

[100] Trousseau and Pidoux, Traité de Thérapeutique.

to converse intelligently, but a paroxysm may be caused by any unusual jar, such as the shutting of a door, or a movement, or even by an attempt to swallow food or drink. The excessive bitter taste, the rapidity of the onset of the symptoms, the rapid progress of the case to a fatal termination or to recovery, the limited number of convulsions, and the normal condition of the intellect during the interval between the convulsions, are the characteristic clinical features of a case of poisoning by nux vomica or strychnin.

The rapidity with which the symptoms of poisoning may supervene will depend, in addition to the conditions above related, upon the method by which the poison is introduced. Absorption rapidly follows the introduction by rectal injection, by insufflation into the air passages, by contact with interior surfaces,—the serous membranes, for instance,—and by subcutaneous cellular tissue; but the most rapid absorption takes place when strychnin is injected into a vein, symptoms appearing in one third of the time required for its absorption from the stomach. Absorption may also take place from the bladder as rapidly as from the stomach.[1] According to Shuler, the mucous surface of the eye is equally active in power of absorption. One to three grains of strychnin placed upon the inner angle of the eye, and left there, will rapidly destroy the life of a man. As this poisonous agent is eliminated with the urine, its presence should be detected in that secretion by chemical analysis. According to Moller's experimental researches,[2] strychnin may be detected within three minutes in the saliva of an animal, which has received a subcutaneous dose of this poison, while in fifteen minutes all proof of its presence in that secretion may disappear. These experiments were confirmed in certain patients into whose subcutaneous tissue Moller injected about 1/10 of a grain; but, according to Dragendorff, it is slowly eliminated, in dogs not until several days after its absorption. If this be true with men, it would not be safe to gradually increase or even continue the doses usually considered within the range of medicinal usage, and which the daily experience of medical practitioners shows to be absolutely safe. Leube and Rosenthal oppose these statements of Dragendorff. Recent experiments of Kratter[3] show that, after the subcutaneous use of 1/10 of a grain (0.0064 gm.) of strychnin, this alkaloid is found in the urine, on an average within an hour, and its elimination ceases within forty-

[1] Segalas, Jour. de Magendie, 1822.

[2] Ugesk. for Läger, R. 3, Bd. XIX, p. 161; quoted in Revue des Sciences Médicales, 1876, t. VIII, p. 477.

[3] Untersuchungen über die Abscheidung von Strychnin durch den Harn, in Wiener Med. Wochensch. 1882, p. 214.

eight hours. Strychnin may be absorbed by the stomach within ten minutes.

A case of accidental poisoning with strychnin has been described by Mr. Bennett,[4] the quantity taken was about 1½ grains (0.1 gm.) in solution. The patient when first seen, which was about an hour after the poison had been taken, was in a rigid and trembling state, and the face almost maniacal in its expression. This was soon followed by a violent tetanic convulsion. Between the fits she did not utter any expression of alarm, but would occasionally request a little cold water. The muscles of the jaws remained so rigid between the spasms that the attempt to introduce the stomach pump was unsuccessful; and, although some strong emetics were got down, it repeatedly happened that the attempt of the patient to take liquids was followed by so violent a spasmodic fit as to prevent her swallowing them, and to give that apparent dread of water so well marked in cases of hydrophobia. During the tetanic fits, the whole body was stiffened and straightened; the neck violently drawn back, the chest fixed, the eyes protruding from their sockets in a horrible manner, the legs pushed out and widely separated, the muscles of the face convulsed, pulse imperceptible, and no breathing could be perceived; the face was livid, more particularly the lips, and froth issued from the mouth. The pupil was also dilated during the paroxysm. It was impossible to produce any relaxation of the body during a fit, and, if moved, the whole body maintained its rigid condition. As soon as death had taken place, which was in an hour and a half, the limbs relaxed, the face and lips gradually lost their livid hue, and became, as well as the body, extremely pallid.

In this case alarming symptoms did not arise until about an hour after the poison had been taken, and numerous cases might be referred to in which an interval of an hour or two occurred. The poisonous effects of strychnin are more rapidly developed than those of nux vomica. But usually the former supervene very speedily, being seldom delayed more than fifteen minutes. Death may occur in twenty minutes, as in the case of Dr. Warner, who was supposed to have taken only ½ grain (0.032 gm.);[5] or in half an hour, as in a case reported by Dr. Theinhart, where 30 grains (2 gm.) of strychnin were swallowed; it is seldom postponed for more than two hours, if no measures for the removal of the poison have been taken.[6] In a case that occurred near London, a prescription was improperly prepared,

[4] Lancet, 1850, Vol. II. p. 462.
[5] Phila. Med. Exam. Oct. 1847, p. 309.
[6] Am. Jour. of Med. Sci. Jan. 1848, p. 303, from Gaz. Médicale.

so that the young man, for whom it was directed, took 1½ grains (0.1 gm.) of nux vomica, and the same quantity of strychnin. It is stated that "he soon afterwards complained of some extraordinary sensations, and almost immediately expired."[7]

It is hardly worth while to enumerate the symptoms of poisoning in the many-recorded cases, reference to which is made in the footnote.[8]

748. **Poisoning by strychnin compared with tetanus.**—It is not always an easy matter for a physician to distinguish between the effects of poisoning by strychnin and a disease called tetanus. A striking symptom in poisoning by strychnin consists in the sudden appearance of the convulsive spasms, and the rapidity with which the attacks and remissions succeed each other, and the short space of time which intervenes between the access of the spasms and a fatal result. We may also mention that the intervals between the spasms are marked by an absolute calm. In tetanus the rigidity of the affected parts is generally permanent, and the access, which is more prolonged than in poisoning, has more the character of a paroxysm and of exacerbation rather than a succession of attacks. Moreover, the fatal result never happens in tetanus in two or three hours after the access of spasms as has been noticed in strychnin poisoning. In the former the duration is generally from two to ten days, and in every case exceeds that of strychnin poisoning, if death supervenes.[9] In tetanus, some cause for the disease, such as a wound, can almost always be detected. The fact that in tetanus the symptoms

[7] Am. Jour. of Med. Sci. April, 1854, p. 537, from Pharm. Jour. July, 1852.

[8] Caussé et Bergeron, Contribution à l'étude de l'Empoisonnement par strychnin, suivie de l'exposé de l'affaire Toulza, dit Rapala, et de la discussion Medico-Légale dont elle a Eté l'Objet, Ann. d'Hygiène, Paris, 1878, pp. 272, 314. Bridges, Poisoning by strychnin, Chicago Med. Jour. and Exam. 1879, p. 48. Rowbotham, Strychnin poisoning, Med. Times and Gazette, London, 1879, I. p. 591. Chaplain, A case of strychnin poisoning, Med. and Surg. Reporter, Phila. 1879, p. 196. *Ibid.* p. 525. Arantham, Case of strychnin poisoning, Southern Clinic, Richmond 1878, p. 1427. Beates, Poisoning by nux vomica, Med. Gaz. N. Y. 1880, p. 34. Jackson, Fatal case of poisoning by strychnin, Australian Med. Jour. Melbourne, 1889, II. p. 54. Foucart, Empoisonnement par gouttes ameres de Baume (made by digesting St. Ignatius beans in alcohol, about five parts in ten). France Médicale, 1880, p. 498. Richet, D'une mode d'asphyxie dans l'Empoisonnement par la strychnin. Acad. des Sciences, Paris, 1880, p. 443; Bos. Med. and Surg. Jour. July, 1877, p. 43. Kelly, A case of strychnin poisoning, Nashville Med. and Surg. Jour. 1882, p. 26. Harrison, A case of poisoning illustrating the antagonism between strychnin and morphin, Lancet, 1882, p. 185. Lefort, De la substitution accidentelle de la strychnin à la santonin dans la pharmacie, Bull. de l'Acad. de Méd. Paris, 1881, p. 460. Delaunay, Influence de la nutrition sur l'Empoisonnement par la strychnin. Comptes Rend. de l'Acad. des Sci. Paris, 1881, p. 432.

[9] *Vide* an excellent account in Compendium de Chirurgie Pratique, A. Bérard et Denonvilliers, Paris, 1841, t. Ier, 350.

progress slowly, and several hours elapse before a severe convulsion occurs, while in strychnin poisoning usually less than one hour elapses; that in tetanus the jaw is the first to be affected, and lock-jaw is constant, while in strychnin poisoning, in those cases in which any difference at all can be perceived, the jaw is the last to be affected; that the duration of the symptoms is numbered by days, in tetanus, and by minutes or hours, in strychnin poisoning; and that the number of convulsions in strychnin poisoning is limited to four or five,—will usually enable the physician to distinguish between the two conditions without difficulty.

749. Poisoning by strychnin compared with puerperal convulsions.—It rarely happens that a pregnant woman becomes the victim of criminal poisoning by strychnin; when such a case does occur, the question as to whether the convulsions were due to the pregnant condition or to strychnin poisoning is sure to arise. The writer is familiar with two cases in which this question arose during the criminal trial, the Hersey case,[10] and the Major case (see Appendix). The following are important points to bear in mind in making a differential diagnosis between the two conditions: 1. When due to the puerperal condition the number of convulsions is usually more than five. 2. In puerperal convulsions, the patient has no recollection of what has occurred, either during the convulsion, or during the interval, the entire period during which the convulsions took place being a blank; whereas, in strychnin poisoning the intellect is intact, except, perhaps, during the last interval and convulsion. 3. Death usually takes place much more quickly after the first convulsion in strychnin poisoning than in puerperal convulsions or eclampsia. 4. When death occurs as a result of puerperal convulsions, it very rarely takes place before the birth or delivery of the child (only a very few cases having been recorded); whereas, in strychnin poisoning this event makes no difference, but in the two cases with which the writer is familiar, death occurred before the birth of the child. 5. In case of death due to puerperal convulsions no strychnin would be detected by chemical analysis.

750. Poisoning by strychnin compared with epilepsy.—As a rule, this question would never arise because the previous medical history of the patient would be known, and because recovery almost always takes place in the case of epileptic patients who are able to be away

[10] Case of Geo. C. Hersey, indicted for the murder of Betsy F. Tirrell; Boston. A. Williams & Co. 1862.

from home without an attendant, if a convulsion happens to occur at such a time. In case, however, that death does occur during an epileptic paroxysm, the fact that only one convulsion was observed and no lucid intervals will enable the physician to eliminate strychnin poisoning as the cause of death; if no distinct history of the convulsion can be obtained, the failure to detect strychnin in the body after death will eliminate it.

751. **Lethal dose.**—The smallest quantity of nux vomica which is known to have caused death is said to be 3 grains (0.2 gm.) of the alcoholic extract; but it is quite uncertain to how much of the powder this corresponds. Hoffman (quoted by Christison) states that two doses of the powder, of 15 grains (1 gm.) each, proved fatal, and other cases are related in which 50 and 60 grains (3.25 and 3.90 gm.) produced death. The smallest quantity of strychnin which has proved fatal appears to be 1 grain (0.065 gm.).[11] In Mr. Bennett's case, above quoted, about one and a half grains were taken.

Recovery occasionally is observed, even after very large doses. Dr. Thomas Anderson records a case in which 7 grains (0.45 gm.) of strychnin were taken without producing fatal consequences.[12] The comparative mildness of the symptoms, and the recovery throw some doubt upon the purity of the strychnin which was used. Dr. Dresbach, of Ohio, attended a man who drank, by mistake, 3 ounces (95 c.c.) of a solution of strychnin, containing 1 grain (0.065 gm.) to the ounce (31 gm.). When seen by Dr. D., about twenty minutes afterwards, he was in the following condition: The whole muscular system rigid, the muscles of the back and legs so rigidly contracted that it was with extreme difficulty that the man was able to walk, face drawn awry and articulation impeded, a sense of burning about the stomach, tightness about the chest, vertigo, dimness of vision, lower extremities cold, and perspiration abundant. Chloroform being the only article at hand which seemed likely to be useful, Dr. D. gave the patient at once 2 drams (7.75 c. c.), and in less than fifteen minutes the relief, Dr. D. says, was complete.[13] A case, in which a man recovered after swallowing 1½ grains (0.1 gm.), is reported by Mr. Foster.[14] In another case a girl swallowed 2 grains (0.130 gm.) of strychnin upon an empty stomach. The poison remained in the stomach fifty minutes before it was removed by an emetic and the stomach pump. The disturbance was slight, and the girl recovered. Dr. Taylor suggests that, owing to the symptoms having been very

[11] Med. Times and Gaz. April, 1855.
[12] Am. Jour. of Med. Sci. April, 1848, p. 562.
[13] Am. Jour. of Med. Sci. Apr. 1850, p. 546, from Western Lancet.
[14] Lancet, 1852, Vol. II. p. 198.

slight in this case, the strychnin was probably not pure.[15] But recently other cases of recovery from still larger doses have occurred, so that it seems more probable in these cases that the absorption was delayed. In one, 4 grains (0.26 gm.) were taken by mistake. Copious vomiting was produced by emetics in about a quarter of an hour, but the system was violently affected, there being not only excessive tetanic rigidity of the muscles, but frequently recurring convulsions, with other symptoms already detailed. The man recovered entirely in two or three days.[16] In another case, by Mr. Chippendale, a man, who had been in the habit of using small doses for an imaginary spermatorrhea, took 4 grains (0.26 gm.) of strychnin and 4 of morphin in 1 ounce of spirit, with the intention of destroying himself. Tetanic spasms ensued in about half an hour ; but in this, as in the preceding cases, the intellect remained unaffected — a fact the more remarkable on account of the large quantity of morphin which had been swallowed. The latter produced none of its peculiar effects, except, perhaps, an itching of the skin, which occurred in the convalescence, and might perhaps be ascribed to it. The man recovered perfectly. The stomach pump was used one hour after the poison had been taken, and water and animal charcoal were injected into the stomach.[17] A case is reported by Dr. Bly, of a man who, by mistake, took 4 grains (0.26 gm.) of strychnin at a dose. Tartarized antimony was immediately administered, but ineffectually, and tetanic convulsions and lockjaw succeeded. These symptoms were palliated by chloroform inhalations, so that an additional dose of tartar emetic could be administered. In thirty-five minutes after the poison had been swallowed the emetic operated freely, and the anesthetic inhalations having been continued, the threatening symptoms ceased in the course of seven or eight hours.[18] In this case it is probable that the tartar emetic, taken immediately after the poison, hindered the absorption of the latter. Mr. Iliff has reported a case in which a female recovered after swallowing two drams (7.75 gm.) of powdered nux vomica.[19]

The attention of the reader is again called to the question of compound poisoning, which has been previously discussed (§ 46). The peculiar action of strychnin, being exerted upon the spinal cord and

[15] Med. Times and Gaz. April, 1854.
[16] *Ibid.* April, 1855. G. Hinnell.
[17] *Ibdi.*
[18] N. Y. Jour. of Med. Nov. 1859, p. 422.
[19] Lancet, Dec. 15, 1849.

the voluntary muscles, will of course be antagonized by other energetic drugs, which have a more or less paralyzing action upon this nervous center, such, for instance, as chloroform, chloral hydrate, morphin, and belladonna; and yet it will be gathered from what has been said in reference to the rapid and energetic action of strychnin, that these antagonists, whose absorption is slower, will not be likely to control the symptoms of its poisonous action, unless previously or simultaneously administered, except in the case of chloroform. Since death is caused by the continued contraction of the thoracic muscles, producing asphyxia, provided this latter effect be prevented by an antagonistic drug for a sufficient time after the administration of strychnin to allow poison to be eliminated by the kidneys, it will be reasonable to assume that fatal strychnin poisoning will be averted. In this connection what has been mentioned in regard to the elimination of the poisonous agent becomes of the highest importance, *viz.*, that the total elimination usually is accomplished within forty-eight hours, and begins within the first hour.

The usual fatal dose as recognized by toxicologists is ½ grain (0.032 gm.) of strychnin or one of its salts. One quarter of a grain (0.016 gm.) is recorded as having caused the death of an adult of thirty-six years within one hundred and five minutes. The symptoms usually begin within five to twenty minutes. Kobert states the limit for a lethal dose for adults as from ½ to 1¾ grains (0.032 to 0.11 gm.), and for children 1/16 of a grain (0.004 gm.).

The subcutaneous injection of .7 of a grain (0.045 gm.), according to the authority of Blyth, will probably cause death. The administration by the mouth would be complicated, if other drugs were taken with it. Schauenstein [20] relates a case of a pharmacist who took, for the purpose of suicide, 7.4 to 9.25 grains (.48 to .6 gm.) of nitrate of strychnin dissolved in 30 cubic centimeters of bitter almond water, and then, after half an hour, 9.25 grains (.6 gm.) of acetate of morphin. He could walk staggeringly for ten minutes afterwards, and then lay on his face in bed, with the pillow case saturated with chloroform, and went to sleep; two and a quarter hours after, somebody entered his room, and the druggist awoke with a very strong convulsion, suspension of breathing, and loss of consciousness. Several convulsions following, the physician was summoned and found him in a state of general convulsions with, finally, opisthotonos. He was

[20] Maschka's Handbuch from Tschepke; Deutsche Klinik, 1861.

given emetics and, afterwards, tannin and codein. The convulsions then became fewer in number and weaker in character, and on the third day he recovered.

In another case, related by Dr. Macredy,[21] a woman took 1½ grains (nearly 10 cg.) and immediately afterwards 2 ounces (62 c. c.) of laudanum; four hours later an emetic of sulphate of zinc was administered. Strychnin convulsions were first observed by slight clonic convulsive movements of the hands and legs, eight hours after taking the strychnin. She was then given chloral hydrate and the convulsions continued for several hours; afterwards her recovery was speedy and complete.

Another case, similar to this, is related by Dr. Harrison:[22] A man took a packet of "Battle's vermin killer," with about 1½ drams of laudanum mixed with rum, on a fasting stomach; he had been drinking freely, and had eaten no food for days. Fifty minutes elapsed before the usual symptoms appeared, and there was no medical treatment given until four hours after taking the dose; at that time he was given chloral and other remedies; he made a rapid recovery. Here is a case where tissues were saturated with alcohol. (See § 46.)

A very interesting case is presented by the following history, in which the last dose of a mixture produced symptoms of poisoning:—[23] A physician prescribed the following mixture:

Tinct. Strophanthi	dr. i.
Liquor strychni hydrochlorici	dr. iiss.
Sol. bismuthi et pepsin (Richardson's)	oz. iss.
Sp. Ammon. aromat.	
Sp. Chloroformi	āā oz. iss.
Aquæ ad.....	oz. vi.

Ft. Misce

Shake the bottle.

Two teaspoonfuls when the attack threatens, and repeat in an hour if necessary.

"Richardson's liquor bismuth" contains 1/20 of a grain (0.0032 gm.) of strychnin in 1 dram (3.89 c. c.); the mixture was alkaline; it contained 1.7 grains (0.111 gm.) of strychnin and 38.25 minims (2.16 c. c.) of chloroform. The patient, a woman fifty-four years of age, had taken the previous doses with considerable relief; but ten minutes after the last dose, which she described as far more bitter than those she had previously taken, she was seized with

[21] London Lancet, Nov. 1882.
[22] London Lancet, May, 1882.
[23] Pharm. Jour. 1893, p. 799.

the usual symptoms of strychnin poisoning, but recovered after five hours. Mr. Blyth, commenting upon this case, suggests as an explanation that "the mixture was alkaline, so that the strychnin was not in the form of a salt, but in the free state, and was therefore dissolved by the chloroform; the amount of strychnin taken in each dose wholly depended upon whether or not the mixture was shaken; if allowed to repose, the globules of chloroform saturated with strychnin were settled at the bottom, and there formed a stratum rich in strychnin, so that the last dose would certainly contain an excess."

752. **Treatment.**—The washing out of the stomach immediately with the introduction of large amounts of water and emetics; but this is difficult on account of the convulsions interfering with the introduction of the tube. In this case, chloroform could be administered to calm the convulsions and to effect the passage of the tube. Kobert recommends a subcutaneous injection of apomorphin for its emetic action. As the patient is excited to convulsive movements in the presence of strangers, or by a direct draught of air, without noises, he should be kept as quiet as possible; even raising the head or touching the lips may start the convulsive movements; medicines, therefore, should be administered as quickly as possible. The respiratory movements are, of course, much interfered with by the convulsions, due to strychnin; and to prevent asphyxia from muscular spasm, it may be desirable to administer oxygen for inhalation.

753. **Action.**—A soluble salt of strychnin injected subcutaneously will act within a few seconds; when introduced into the punctum lacrymale (in one case), a dose of 5.4 milligrams will produce violent tetanic convulsions in four minutes.

Nux vomica powder, or strychnin in pill form, will not have such a rapid course of absorption, symptoms not generally appearing until from one half to two hours or even longer. The peculiarity of the convulsions shows a condition like that of violent general tetanus: Limbs stretched out involuntarily; the hands clinched; the soles incurved; and, in the height of the paroxysm, the back arched, and rigid as a board, the sufferer resting on head and heels so that the hand can be passed between the two extremities without touching the body; the abdomen tense; the muscles of the walls of the chest are set immovably; and the face becomes suffused and reddened, with prominent and staring eyes.

As previously remarked, the convulsions and remissions, or intervals between, recur until death or recovery; and, as a rule, within two hours from the commencement of the symptoms, the case in some

way or other terminates; the duration of a spasm may vary from thirty seconds to five or even eight minutes; the interval between lasting from forty-five seconds to one or even one and a half hours.

754. Diagnosis.—The symptoms of strychnin poisoning, which have been so fully described in the earlier parts of this article, are very distinctive,—especially those which refer to strychnin convulsions. A confusion may arise in distinguishing these convulsions from those of tetanus and chorea, from puerperal and uremic convulsions, and from hysteria. The only similarity in the convulsions of the above diseases is that of violent tonic spasms; in other respects, the symptoms are unlike. The hysteria in a traumatic tetanus will show a previous injury as the cause of lockjaw and the disease will usually come on gradually, and progress slowly to a fatal termination during a period of several days; in strychnin poisoning there is no antecedent injury, and the manifestations of the poisoning appear suddenly, and progress rapidly to a fatal termination; in tetanus, the first muscles to be convulsed are generally those of the neck and of the jaw, with a persistent lockjaw, while, in strychnin poisoning, the muscles of the extremities are first affected and those of the neck and jaw last.

As remarked in the previous pages in the description of the symptoms, it is especially noted that there is almost complete relaxation of the muscles between the convulsive movements and the opisthotonos. A competent medical expert should be able to distinguish between the diseases tetanus, hysteria, etc., and the peculiar strychnin set of symptoms so carefully described in the earlier pages of this article. (See *supra*, §§ 748, 749, 750.)

755. Post-mortem appearances.—The post-mortem appearances are by no means characteristic, though certain negative results can be drawn therefrom, which are not without value when considered in relation to the symptoms observed during life. It may be difficult to prove whether the pathological peculiarities found at the autopsy of persons poisoned by strychnin may not have been due to some other condition. No notice should be taken of those cases in which there is no appreciable trace of pathological alteration of the organs, though this absence of lesion is not without value.[24] The most significant pathological lesions would be found in the nervous centers; but it must also be remembered that there are certain diseases in which identical changes have been observed. (Abercrombie.)

To some extent the degree of rigor mortis of the body, and the per-

[24] Tardieu, op. cit. p. 940.

manence of this condition, are peculiar. Reference is asked to what has been mentioned in a previous chapter on this subject (§ 26). Until the researches of Brown-Séquard and others during the last few years, it was supposed that this was a pathognomonic condition; but it seems now to be pretty well established that the condition of cadaveric rigidity depends upon a certain peculiarity of the muscular system, and that, when death has followed the use of convulsive poisons, the early and prolonged condition of the rigor mortis is due to the effects of the convulsions upon the muscular system. Now it is evident that, in any case of death which has been preceded by violent muscular movements, this same early appearance of cadaveric rigidity may occur; while, on the contrary, if the convulsions have been prevented by the use of medicaments, or perhaps by artificial respiration,[25] this post-mortem peculiarity may be absent.[26] In the case of Cook, poisoned by Palmer, Dr. Harland testified that the body was very stiff, more so than dead bodies generally are. The muscles were very highly developed; they were strongly contracted and thrown out. The hands were firmly closed.[27] In another case, reported by Mr. Wilkins, it is stated that seven hours after death the rigidity of the body was so great as to allow it to be lifted by the heels; it is described as being "as stiff as wood."[28] In some cases there have been found signs of inflammation in the intestinal canal, and very generally congestion of the brain and its membranes, and sometimes softening of its substance and of the spinal marrow. The right cavities of the heart are usually contracted, and the blood dark and fluid. In some instances, no doubt, these appearances were due to cadaveric changes, and were not the result of any peculiar influence of the poison. In the Major case (see Appendix) the cadaveric rigidity was very marked two weeks after death, when the body was exhumed by the writer. If death takes place during a convulsion, as is commonly the case, while the action of the respiratory muscles is suspended, there will be found a venous congestion in all parts of the body; but this appearance is not, of course, characteristic of strychnin poisoning.

X. Opium and its alkaloids.

756. Occurrence.—Opium is the dried juice of the Papaver somnif-

[25] *Vide* an article on Hydrocyanic Acid, in London Practitioner for April, 1872.

[26] Croonian Lecture, by Prof. Brown-Séquard, in Proceedings of the Royal Society, May 16, 1861. Also Am. Jour. of Med. Sci. Jan. 1870, pp. 87, etc.

[27] Times and Gazette, May, 1856.

[28] Guy's Hospital Reports, 3d ser. III. 484.

erum, a poppy which is grown chiefly in China, India, Asia Minor, and Egypt, but has been cultivated to a certain extent in colder climates.

The crude drug, opium, contains various active principles, such as morphin, 12 to 16 per cent; codein, 0.03 per cent; narcein, 0.02 to 1 per cent; narcotin, 1.30 to 11 per cent; thebain, 1 per cent; papaverin, 1 per cent; meconic acid, 3 to 4.3 per cent; and traces of other less important constituents, such as cryptopin, hydrocotarnin, pseudo-morphin, laudanin, protopin, meconidin, laudanosin, lanthopin, and rhœadin. Of these alkaloids, morphin, codein, narcotin, thebain, narcein, and papaverin are poisonous to the lower animals; but there are no recorded instances of death being caused in man by any other than the morphin. As morphin represents the principle of opium which is poisonous to man, we shall discuss the action of this alkaloid rather than the crude substance imported under the name of opium.

757. Properties and tests.—Pure morphin crystallizes in the form of prisms belonging to the rhombic system. It is soluble in about 1,000 parts of cold water, and in 500 parts of boiling water; also in about 100 parts of cold alcohol and in 36 parts of boiling alcohol; it is almost insoluble in ether, and but slightly soluble in chloroform; it is very soluble in amyl alcohol; also very soluble in dilute acids; acid salts of morphin, which are readily soluble in both water and alcohol, are thus formed.

The following are some of the chief characteristics of morphin:

1. Neutral sesquichlorid of iron, dropped on crystals of morphin, renders them blue, or, added to an aqueous solution of morphin, gives a blue color, unless an excess of the iron compound be added, in which case a green color is produced; therefore, the precaution should be taken to add, at first, only the smallest possible amount of the iron solution. It is usually stated that this green color is due to an excess of acid in the iron solution; but the ordinary laboratory reagent, which always contains a slight excess of acid, will give the blue color if not added in excess; as the writer has repeatedly observed. This is an exceedingly delicate test, when the morphin is pure; but the presence of strychnin, or other organic matter of various kinds diminishes its delicacy. The solution of morphin should not be more dilute than 1 : 5,000. This test is also valuable, because the mixture of iron and morphin solutions does not prevent the reaction with nitric acid (see 4). Fallacies.—Tannic and gallic acids with a little water, and an infusion of cloves or pimento, also form blue compounds with sesquichlorid of iron.

2. Fröhde's reagent, which is concentrated sulphuric acid containing in solution molybdate of sodium or ammonium in the proportion of 1 centigram of molybdate in 1 cubic centimeter of sulphuric acid, forms one of the most delicate tests for morphin which we have. A drop of this reagent, brought in contact with morphin or one of its salts, dissolves it, producing immediately a beautiful violet-colored solution, which changes soon to a green, then to a brown-green, later to a yellow, and after twenty-four hours to a bluish-violet again. This is an exceedingly delicate test, reacting with morphin when only .05 of a milligram of the latter is tested. (Dragendorff.) This same reagent gives characteristic color reactions with many of the other alkaloids.

3. Husemann's test[29] is also an exceedingly delicate one for morphin. It is performed as follows: Allow a crystal of morphin to dissolve in a drop of concentrated sulphuric acid and remain for twelve or fifteen hours at the ordinary temperature, or for one half an hour at the temperature of boiling water, for an instant at 150° C. (302° F.), and then introduce a small particle of some oxidizing agent, such as nitric acid, niter, chlorate of potassium, chlorin water, sodium hypochlorite, or sesquichlorid of iron, when a beautiful blue or reddish-violet color is produced, which changes quickly to a blood red, and gradually becomes colorless. This test is delicate for .01 of a milligram of morphin. Other organic substances do not usually interfere with it, if one of the chlorin compounds is used as the oxidizing agent.

4. Nitric acid reddens morphin or its salts (the chlorate excepted, according to Dumas), and forms with them an orange-red solution, which is much darkened by an excess of ammonia, and which becomes yellow after a little time. Fallacies.—Nitric acid produces a red color with several other bodies, as brucin, commercial strychnin, several volatile oils (as oil of pimento and oil of cloves), some resinous substances, etc.

5. Iodic acid is deoxidized by morphin, iodin being set free. Hence, when this alkaloid is added to a solution of iodic acid, the liquid becomes reddish-brown, and forms a blue compound with starch (iodid of starch). Fallacies.—Sulphureted hydrogen, sulphurous acid, phosphorous acid, sulphocyanid of potassium, sulphosinapisin, and all other reducing agents, have a similar effect on iodic acid. Of course if the morphin be pure, these fallacies have no application.

[29] Fresenius's Zeitschrift, 1876, p. 103.

6. Morphin is precipitated from solutions of its salts by phosphomolybdic acid. This reagent being very sensitive, the process of preparing it is here given: A yellow precipitate from molybdate of ammonium, to which nitric acid has been added, is obtained by phosphate of sodium, and well washed; this precipitate is then suspended in water, and heated with carbonate of sodium till perfectly dissolved; then the solution is evaporated to dryness and calcined to drive off the ammonia. If, after the calcination, only a partial reduction has been accomplished, the mass is again calcined, after having been sprinkled with nitric acid. Afterwards heat the calcined mass in distilled water, add enough nitric acid to make the solution strongly acid, and a sufficient quantity of water to make 10 parts of the liquid to 1 of the saline substance. In this way a golden-yellow liquid is obtained, which should be kept away from ammoniacal vapors. This reagent with .2 of a cubic centimeter of a solution of sulphate of morphin, of the strength of 1 part in 5,000 of water, gives, gradually, a turbidity. It also precipitates many of the other alkaloids from solutions of their salts.

7. If morphin be mixed with a little cane sugar, and this mixture treated with concentrated sulphuric acid, a wine-red color is produced. (Weppen.) This test will show the color with .01 of a milligram.

8. Among other tests for morphin the following may be mentioned as the more important: 1. Morphin is precipitated from solutions of its salts, when not too dilute, by ammonia water; and the crystals of morphin thus obtained may be examined with the microscope. 2. A crystalline precipitate is also produced by Mayer's reagent, which contains, in 1 liter of water, 13.546 grams of corrosive sublimate, and 49.8 grams of iodid of potassium. 3. A solution of iodid of potassium produces, in a solution of a salt of morphin, a precipitate consisting of groups of white, silky needles. 4. A brown precipitate is produced by a solution of iodin in iodid of potassium, similar to those given by this reagent with many of the other alkaloids. 5. A solution of bichromate of potassium gives, with solutions of morphin, an amorphous precipitate, which gradually becomes crystalline. All of these precipitates may be formed in a drop of the solution of morphin in a watch glass or upon a glass slide, by the addition of a drop of the reagent; and the precipitate can then be easily examined with the microscope.

758. Preparations.—The present United States Pharmacopœia (1890) describes a deodorized opium, in the form of 100 grams of

powdered opium, which shall contain 13 to 15 per cent of morphin, by macerating the same with ether, and adding recently dried sugar of milk in powdered form in sufficient quantity to make 100 grams. This is evidently a substitute for the requirements by the edition of 1880, that the maximum strength of opium shall contain 16 per cent of morphin; and this obviates the necessity of cautioning the medical public, as we mentioned in our previous edition, against the strength of the opium preparations of the Pharmacopœia in the edition of 1880. The preparations of opium prescribed by the edition of United States Pharmacopœia (1890) are now of uniform strength, based on the required presence in opium of nine per cent of morphin, except in those preparations where the deodorized opium is prescribed for use.

Other preparations (U. S. P.) are opium and ipecac powder; glycyrrhiza troches; opium and ipecac tincture; deodorized tincture of opium; extract of opium containing 18 per cent of morphin—the process of assay described; opium pills, 100 pills containing 100 grains (6.5 gm.) of powdered opium; opium plaster containing 6 per cent of extract of opium; tincture of opium containing 10 per cent of powdered opium, 100 cubic centimeters of this tincture should yield 1.3 to 1.5 grams of crystallized morphin; camphorated tincture of opium (paregoric) containing 4 grams of powdered opium in 1,000 cubic centimeters; vinegar of opium containing 100 grams of powdered opium in 1,000 cubic centimeters; wine of opium, of same strength as preceding vinegar of opium.

759. **Absorption into system.**—Opium itself is absorbed into the system slowly or rapidly, according to the method by which it may be administered, and the form in which it is given; for instance, an old opium pill may be swallowed and so slowly acted upon by the digestive fluids in the stomach, that its peculiar effects follow very slowly. Opium may be administered, combined with oil of theobroma, as a suppository in the rectum, and be slowly absorbed. Liquid preparations of opium are absorbed more slowly in the rectum than when swallowed into the stomach. Opium administered to a fasting stomach is more rapidly absorbed than when taken with food. Upon these and similar circumstances depend the absorption of opium and its active principle, morphin; hence it follows that when it is more slowly absorbed, it may be gradually undergoing elimination from the body, and the symptoms of its action may be delayed, or so much retarded and modified that what might in one case be a poisonous dose becomes under other circumstances a nonpoisonous

dose. In regard to its action in diseased states of the eliminating organs, reference is made to a paragraph (§ 4), where this part of our subject is fully discussed.

Opium smoking is another method of its administration, and though not precisely like the effects produced upon the body by other avenues of introduction, its stupefying and intoxicating effects are pretty generally known from the popular works of De Quincy and other writers; in the case of smoking, however, some of the alkaloids are destroyed or weakened by the action of heat or by combustion, and other injurious alkaloids formed, such as picolin.

The tincture of opium in proportionate doses causes nearly the same symptoms in man as may follow the use of morphin, though it is less liable to cause the troublesome itching or pruritus of the skin observed after morphin has been absorbed into the system.

760. Morphin and its relative alkaloids.—The most important alkaloids of opium, in reference to the poisonous action of this drug, are morphin, codein, narcotin, narcein, papaverin, and thebain. Of these, morphin is by far the most important alkaloid to which opium mainly owes its toxic characteristics. Morphin is found in opium combined in part with meconic acid, and in part with sulphuric acid. In toxicology, morphin and meconic acid are the two most important constituents of opium, because, in the analyses for the detection of cases of poisoning by opium, these two bodies are usually separated and identified. Meconic acid is not found in any other substance except opium, and, consequently, when detected, is generally sufficient to furnish the indication of the presence of opium.

761. Symptoms of acute poisoning.—No matter in what way morphin may be given to a person in good health, the absorption of an amount sufficient to produce poisonous effects is followed by a train of symptoms easily recognized under the head of opium narcosis: A sensation of fullness or dizziness of the head, accompanied by a feeling of mental and physical ease, an exhilaration of intellectual activity, and a rather rapid pulse; often, however, there is an unpleasant, disquieting sensation of an intolerable itching of the skin, pruritus,—especially noticed about the nose and forehead,—which the patient makes slow and feeble efforts to relieve by scratching, and which may extend all over the body, and may even interfere with the sleep-producing effects of the drug. Nausea and vomiting may be an early symptom; and, sometimes, when the dose has been too large, this symptom is so severe that it may cause rejection of a large amount of the poison, and thus prevent the absorption of an

amount sufficient to cause death; it is in this way that, occasionally, a person intent on self-destruction has awakened to a consciousness of his object having been defeated. Faintness is sometimes an early symptom, but instances of this are not common.

The symptoms produced by a poisonous dose of opium or its preparations differ from those which are occasioned by moderate and remedial doses of the drug. While, in the latter the purely narcotic effects do not occur without a certain degree of previous exhilaration and stimulation, in the former, symptoms of mental depression come on, such as lassitude, headache, a peculiar oppressive feeling in the head, drowsiness, stupor, and complete insensibility; this last condition may pass on to coma, and is generally preceded by stertorous breathing. During this stage, reflex action to external noises, and the irritation of the skin, are exaggerated; but the patient can be roused, though he immediately relapses into a state of stupor. During coma, the patient cannot be roused by any noise. The respiratory movements are peculiarly recognized under the name of "narcotism," being at first hurried, and afterwards labored and irregular and slow; the breathing may become more and more difficult and slow and irregular, as described under the name of the "Cheyne-Stokes type." During the next period, the skin is cold and moist, because opium and morphin arrest all secretions except that of the skin, and there is generally relaxation of the muscular contraction; the pupils, in the early stages, are contracted and insensible to light; in the latter stages of opium or morphin poisoning, they may be dilated and also insensible to light, and this latter phase is generally a fatal sign, indicating general muscular paralysis. Though constipation is, as a rule, present, there may be, in some cases, a slight purging action. It is known, generally, that vomiting, occurring before the presence of stupor, is a favorable sign of recovery. The peculiar odor of opium may be noticeable in the breath, and so distinguished from the narcotism produced by alcohol,—especially when taken in the form of a liquid preparation. Poisoning by morphin and its salts is not followed by the smell of opium in the breath. Occasionally, there are idiopathic variations in these symptoms, as, for instance, a high temperature with a quickened pulse, and spasms of a tetanic nature with dilatation of the pupils, may be noticed in the earlier stages. The fatal signs of poisoning in acute morphinism are an extremely feeble pulse; pupils dilated and not responsive to the action of light; generally, muscular relaxation, shown by dropping of the limbs, falling of the lower jaw, and relaxation of the

sphincter muscles; sometimes persons recover from the severe symptoms of opium or morphin poisoning, and then fall suddenly, sometimes even after a period of days, into a relapse, become comatose, and die.

In the police records there may be found cases of somewhat the same symptoms, which are attributed to opium poisoning, but they are really due to a hemorrhage in the pons Varolii (brain). This error is caused on account of the peculiar action of the contracted pupils, and the insensibility to light which exists in that accidental disease. Sometimes carbolic acid poisoning has been mistaken for opium poisoning; as in the former case, the pupils are contracted, and there are strong signs of narcotism. This error should not occur, because, in carbolic acid poisoning, the odor of this acid is very strong. Morphin occasionally produces symptoms, which seem similar to those occurring in strychnin poisoning unless carefully observed; but they should be distinguished from strychnin spasms, since convulsions from these two drugs are distinctly different. We have remarked in our first chapter that, where there are pathological conditions of the organs,—especially of the kidneys (pre-eminently the contracted granular form),—fatal effects of morphin in small doses are more prevalent on account of the slow elimination by the kidneys of this drug. This is an important consideration in medico-legal cases, and the reader is referred to that part of the first chapter in which this subject is more fully treated.

There is a very interesting case in regard to this matter reported in 136 New York Court of Appeals cases, p. 423, 33 N. E. 65, *People* v. *Harris*. In this case the defendant was convicted of murdering a woman by administering morphin; but he insisted that she died of uremic poisoning, from a diseased condition of kidneys, or of a hemorrhage of the pons Varolii.

The court declared that the fact that the deceased was able to speak with clear articulation and rationally when she was awakened from her sleep was significant, and was something which is not possible where a person is attacked with uremic coma or with hemorrhage of the pons Varolii in the brain, while it is quite possible in the first stage of morphin narcosis; and the court further declared that a sense of exhilaration, first noticed from her remark about having had pleasurable dreams, did not comport with any disease of the brain or with uremic poisoning.

In this case it appeared that the deceased had no constitutional idiosyncrasy as to morphin; because, a few months before death, she

had doses of ¼ grain (0.016 gm.) every three hours for about twelve hours, and of 1/6 grain (0.011 gm.) thereafter every four hours for twenty-four hours.

It appeared that a minimum dose to produce the conditions narrated of the deceased would be, according to the evidence, from 2 to 3 grains (0.13 to 0.19 gm.) of morphin.

The following is in the opinion of the court per Gray, J.:

"Great bulk of this record is made up of the evidence given on behalf of the People to establish a poisoning by morphin as the cause of the death, and to demonstrate the guilt of the accused; while the evidence in his behalf was confined to the examination of several medical experts, for the purpose of proving that the death of the deceased might be attributed to other causes than to morphin poisoning. The evidence connecting the accused with the commission of the crime charged was wholly circumstantial. There was neither testimony by some eyewitness of the giving of the poison, nor of any admission by the accused." Body of Helen Potts (victim) was exhumed fifty-five days after death from a brick vault, where dry, gravelly soil kept the air in vault dry; the body had been embalmed, and was in good state of preservation (girl nineteen years of age).

There was no appearance at autopsy of any evidence of disease of heart or kidneys, or of pregnancy. Brain congested as usual in opium poisoning. Stomach removed, submitted to a chemist, who found morphin in its contents and also lining membrane, and no evidence of quinin (as claimed by convicted he had given her). The fluid used for embalming contained no morphin.

A sufficiently large dose, or the continued use of several small doses, causes a very marked contraction of the pupil of the eye, and generally a loss of sensitiveness of the surface of the eyeball. The experienced medical practitioner will recognize this condition of the eye as peculiar to opium narcosis. After these primary symptoms, the individual will fall into a condition of sleep or drowsiness accompanied with quiet and pleasant dreams, not unfrequently with a cold and clammy skin bathed in perspiration, with suppression or retention of urine, and sometimes with increase of surface temperature and a general blush. If the dose be repeated after this train of symptoms has appeared, or if the original dose has been large enough, these symptoms grow more intense, and the pulse and respiratory movements become very much slower, the latter being reduced from the normal number of 18 per minute to 3 or 4 per minute. With poisonous doses, the period of intellectual excitement and exhilara-

tion is almost always so short as to escape observation, and the whole train of symptoms above described in detail follow each other so rapidly that the victim may fall into a deep sleep from which he may at first be aroused, but this is soon succeeded by coma, and stertorous and slow breathing, the pulse is sluggish and scarcely perceptible, the face dusky and cyanotic, the extremities cold and clammy to the touch, the pupils contracted to a pin's point; but just before death takes place, signs of asphyxia are present, the pupils dilate, and the pulse becomes quick and scarcely perceptible. It should be noted here that the urine may be albuminous and contain casts from the tubules of the kidneys, which may lead to the suspicion that the victim is the subject of Bright's disease. We have already seen in fatal poisoning by other violent poisons (mercury, ammonia, arsenic, phosphorus, etc.) that this eliminating organ may be subjected to such efforts to get rid of the fatal drug that it becomes congested, and may show signs of disturbance of its functions by secreting albuminous urine containing casts; this is undoubtedly a common occurrence in poisoning, and should not be overlooked. If the kidneys have been the subject of disease, the post-mortem appearances will solve the doubt whether the disease or the drug is responsible for the fatal result.

The apparent inconsistency in the presence of the two opposing conditions of dryness and excessive moisture of the surface of the body needs some comment. Generally speaking, the dryness and warmth of the surface of the body is the most common symptom, but there are a number of undoubted cases of morphin poisoning where copious perspiration is a singular and unfrequent symptom. It is mentioned by Christison, who says that in one case "the sheets were completely soaked to a considerable distance around the body;" and Dr. Morland observed it, in an equal degree, in a case he has reported.[30] Delirium is very rare, and, when it occurs, is of a passive character. In the adult, convulsions have been seldom observed, although one or two curious cases have been reported in which they were witnessed. In children, however, they are not uncommon. Occasionally, spontaneous vomiting takes place,—especially after the ingestion of the drug in large quantities,—and some instances have occurred in which this early rejection of the poison from the stomach has saved the life of the individual. A child nine years of age, mentioned by Dr. Coale, recovered in this way after having swallowed 4 grains (0.26 gm.) of opium and 4 of extract of belladon-

[30] Am. Jour. of Med. Sci. Oct. 1854.

na. The pulse also varies in character, being usually feeble and irregular, but sometimes full and slow; in a case reported by Dr. J. B. S. Jackson, it is described as "rapid, full, and throbbing." Such differences depend often, probably, upon the variable periods at which the observation is made; thus, the skin is warm and perspiring, and the pulse rapid and feeble, or perhaps even strong, early in the case; while later, if the patient gets worse, the surface becomes cold and pale, and the pulse slow, feeble, and irregular. Much also may depend upon the constitutional irritability of the system; but, in persons who have accustomed their system to the use of morphin, enormous doses may be tolerated without producing opium narcosis or other dangerous effects. It should be remarked that the pernicious habit of a continued use of morphin most usually entails disease and an early death from chronic poisoning, though there are exceptional cases.

The differences between poisoning by opium and by other narcotics are briefly these: Aconite, digitalis, and tobacco do not produce stupor, nor does conium, except in very large doses, and even then not uniformly; hyoscyamus, stramonium, and belladonna excite violent delirium, and extreme dilatation of the pupil. Inebriation by alcohol bears a very close resemblance to opium narcotism in many cases; but the former is preceded by confusion of ideas or complete delirium, and the breath is strongly tainted with the alcoholic odor.

The symptoms usually commence, in the adult, within an hour after the poison has been taken; but sometimes the confirmed narcotic effects do not come on until a later period. In a case quoted by Dr. Taylor, the patient was found totally insensible in fifteen minutes. In Dr. Lyman's case,[31] a female, after taking 1 ounce of laudanum with suicidal intent, began very suddenly, in thirty-five minutes, to lose her pulse and muscular power, and had slight spasms; the lips became livid, there was spasmodic dropping of the lower jaw, the extremities were cold, and in ten minutes more she was unmistakably dead. Thus, three quarters of an hour only elapsed from the ingestion of the laudanum to her death. Dr. Coale[32] states that he met with a case fatal in the same time, and Dr. Taylor quotes a similar one. Dr. Beck reports a case which proved fatal in two hours. Another is given which terminated in two and a half hours.[33]

The average duration of cases of poisoning by opium is stated by Christison to be from eleven to twelve hours. The rapidity, or indeed the certainty, of death does not always correspond with or de-

[31] *Ibid.*
[32] Am. Jour. of Med. Sci. 1850, p. 73.
[33] Boston Med. and Surg. Jour. Vol. XI. p. 285.

pend upon the amount taken, when this is beyond the limits of safety. Among the cases above referred to is one by Dr. Jackson, which recovered, although 90 grains (5.83 gm.) of opium were taken, and no relief was afforded for three hours afterwards. In the case mentioned by Dr. Taylor, of a man who had taken from 28 to 30 grains (2 gm.) of opium the symptoms were so little characteristic of poisoning by this drug that no suspicion was entertained of its having been used, and death took place rather suddenly in ten hours after the fatal dose had been swallowed.[34] On the other hand, the most rapidly fatal case yet recorded was the one above referred to, in which only 1 ounce (31 c.c.) of laudanum was swallowed. The fact should, however, be borne in mind that laudanum, or any solution of opium, is more prompt in its effects, because it is more readily absorbed than solid opium.

762. Symptoms of chronic poisoning.—Chronic poisoning by opium, called "morphinism," is quite common. It generally occurs from the original using of morphin—especially by subcutaneous injection—for the relief of pain.

The hypodermic or subcutaneous method of introducing opium into the system is unfortunately well known to others than medical practitioners, and undoubtedly might be used in cases of criminal intent, though no recorded cases are known to the writer where a criminal has resorted to this means of destroying the life of his victim. The pain involved in the method is an obvious disadvantage, which would occur to the mind of an unprofessional person desiring to escape the consequences of his criminal action.

Morphin itself is rarely used, as it is not so soluble in water as its salts. By an easy computation, 1 part of the sulphate and the hydrochlorate of morphin will be found to contain about three quarters of their weight of morphin, and the acetate about two thirds. The most commonly used of these salts of morphin are the sulphate and acetate. The subcutaneous method of introducing morphin is followed in about fifteen minutes with the symptoms of its action; and, in several recorded instances, where this method has been employed upon persons whose great susceptibility to the poisonous action was unknown, a small dose, ¼ grain (0.016 gm.), has caused death, and one instance is recorded where even ⅛ grain (0.008 gm.) was followed by profound and fatal coma, or a state of hebetude from which it was impossible to arouse the patient. Occasionally, too, a care-

[34] Taylor's Med. Jurisprudence, 6th ed. p. 162.

less introduction of the drug directly into a vein has deluged the central circulatory system with a dose which would not, if slowly absorbed, have done any material harm.

763. Lethal dose.—Children are more susceptible to opium poisoning than adults even under the rule of the *pro rata* dose as between infants and grown persons. This susceptibility to poisoning from opium lies especially in its action upon the respiratory nerve centers. Children under five years should not receive a dose over 0.05 to 0.18 grains (0.003 to 0.12 gm.) of morphin. A poisonous dose for adults would be 1 to 3 grains (61 to 184 mg.), in persons not addicted to the use of the drug; among persons habituated to its use very large doses may be taken with impunity, so far as the acute symptoms of poisoning are concerned. These doses refer to the ingestion by the stomach. The subcutaneous use of morphin must be restricted to much smaller doses, on account of the more rapid absorption in the blood at one time. It is always safer to commence with a small dose, say 1/8 or 1/6 of a grain (8 to 10 mg.). This dose should not be repeated for an hour as a medicinal remedy to a person unaccustomed to its use. Dangerous and, sometimes, fatal results have occurred, not frequently, however, after a subcutaneous injection of ¼ of a grain (0.016 gm.).

The pharmacopœias of Germany, Great Britain, Austria, and Switzerland, prescribe the following:

Maximum Medicinal Quantities of Morphin or Its Salts for Adults.

	In one dose.		In 24 hours.	
	Grains.	Grams.	Grains.	Grams.
German Pharmacopœia 1895	0.5	0.03	1.5	0.10
German Pharmacopœia 1872	0.5	0.03	1.9	0.12
British Pharmacopœia 1885	0.5	0.03		
Austrian Pharmacopœia	0.5	0.03	1.9	0.12
Swiss Pharmacopœia 1893	0.5	0.03	1.5	0.10

Maximum Medicinal Quantities of Opium for Adults.

	In one dose.		In 24 hours.	
	Grains.	Grams.	Grains.	Grams.
German Pharmacopœia 1895	2.3	0.15	7.7	0.50
British Pharmacopœia 1885	3.0	0.20		
Austrian Pharmacopœia	2.3	0.15	7.7	0.50
Swiss Pharmacopœia 1893	2.3	0.15	7.5	0.50

Owing to the varying susceptibility of individuals to the poisonous action of opium, it is not possible to state the amount which will be uniformly fatal. The dose which may cause poisonous symptoms

varies even among those unaccustomed to the use of morphin; but when the habit of taking morphin is an old one it is impossible to attach any limit to the tolerated dose. Cases have been reported where a dose of from 20 to 50 grains (1.3 to 3.24 gm.) has not caused a fatal result. It should not be gathered from what is here stated that no dose of morphin can cause death even in those who are habituated to its use, since this inference is not true; the only doubt exists as to stating in advance what number of grains is sufficient to cause death in any given case of a morphin eater, who takes a definite amount *per diem* of the same drug. There are two factors which interfere with a correct answer to this question; first, the difficulty of ascertaining accurately what amount of morphin is being taken each day; and second, the insufficient number of attested cases upon which to base an opinion. The former of these conditions is obscure, from the fact that the opium or morphin eater is especially anxious to conceal his habit, as well as the amount which he takes daily, and the vitiated intellectual condition caused by his habit has so obtunded his moral sense that no intelligent and experienced medical practitioner would accept his statement on oath as to the dose he habitually takes.

The personal experience, not only of the editors, but of other physicians who have had under observation cases of this character, tends to show that it is only by dogging the steps of the individual and those persons about him that any correct surmise can be made. But, leaving entirely out of our consideration of this question the morphin and opium eater, it is difficult to form an opinion of the precise size of the fatal dose of morphin required in individuals not accustomed to its use.

A case is referred to by Dr. Taylor in which 4 grains (0.26 gm.) proved fatal; and another in which death was supposed to have resulted from a dose of 2 drams (7.75 c.c.) of laudanum, but it was uncertain whether as much as half an ounce had not been swallowed. A gentleman affected with acute rheumatism died comatose after taking, at intervals of an hour, four pills, each of which contained ⅓ of a grain (0.022 gm.) of morphin.[35] In Dr. Lyman's case, 1 ounce (31 c.c.) of laudanum was the cause of speedy death. The smallest dose which has proved fatal to a child is 1/20 of a grain (0.0032 gm.) of opium.[36] The child was six days old. Dr. J. B. S. Jackson met with a case in 1845 in which five drops of laudanum, injected into the rectum of a child eighteen months old, caused death in six hours. In-

[35] Med. Times and Gaz. June, 1860, p. 254.

[36] Dr. E. Smith, Assoc. Med. Jour. April, 1854.

stances are quoted by all writers on toxicology of death in children from extremely small doses, such as 1/10 or 1/5 of a grain (0.0065 or 0.013 gm.), and most practitioners have witnessed alarming symptoms from a few drops of laudanum, or fractional doses of Dover's powder given to children. In many instances these have been dissipated only by active medical interference, such as cold affusion, galvanism, etc.[37]

Trousseau states that he has seen narcotic effects in children from a dose of the wine of opium, equivalent to less than 1/100 of a grain (0.0005 gm.) of this drug. It is well known that a child may be narcotized by the milk of a nurse who has taken opium. Bouchardat relates that nine newborn children were narcotized by the decoction of a single poppyhead. In London, an infant four days old was destroyed by 1/20 of a grain (0.0032 gm.) of opium, or about one drop of laudanum;[38] and in Edinburgh the same effect was produced by two drops of laudanum in an infant also four days old.[39]

The following cases prove the possibility of recovery after excessive doses of this drug. A gentleman, seventy-two years of age, recovered from the effects of 12 drams (46.65 gm.) of laudanum;[40] another, aged thirty-five years, after ½ ounce (15.55 gm.) of this preparation had been taken;[41] in the third case, above an ounce was swallowed, and, although the symptoms were intense, the patient recovered, temporarily at least, with paralysis of the right side.[42] Another case is reported, in which 90 grains (5.83 gm.) of opium were taken by a female, who, however, recovered.[43] An infant of twelve months has recovered from the effects of 72 drops of laudanum;[44] another, six days old, after 2 grains (0.13 gm.) of powdered opium;[45] and a child, not quite six years old, from a dose of 7½ grains (0.48 gm.) of opium, which were, however, mixed with an equal quantity of prepared chalk.[46]

According to Dr. Taylor,[47] cases are recorded where 1 grain (0.065

[37] *Vide* I. Young's case and references given by Dr. Hays, Am. Jour. of Med. Sci. April, 1852, p. 426; also a very instructive case by Dr. Herapath, where respiration was artificially maintained by means of the galvanic battery until the narcotism passed off and the child was saved. Lancet, Am. ed. 1852, p. 450.

[38] Times and Gazette, April, 1854, p. 386.

[39] Edin. Med. Jour. II. 146.

[40] Lancet, July, 1857, p. 80.

[41] Boston Med. and Surg. Jour. Aug. 1855, p. 21.

[42] Brit. and For. Med.-Chir. Rev. XXII. 523.

[43] Am. Jour. of Med. Sci. Oct. 1854, p. 385.

[44] Edin. Med. Jour. III. 716.

[45] Boston Med. and Surg. Jour. Dec. 1857, p. 357.

[46] Am. Jour. of Med. Sci. April, 1859, p. 367.

[47] Treatise on Poisons, Phila. 1875, p. 549.

gm.) of the hydrochlorate of morphin has proved fatal to adults, one of whom took the medicine in solution, and died in about seven hours; a second, in pill, and died in thirteen hours; a third, in a powder, and died in about ten hours; and a fourth, by subcutaneous administration, and died in about eighteen or twenty hours. A fifth case[48] is reported in which a woman died in twelve hours after taking only 1 grain (0.065 gm.) of morphin in a pill. "Dr. Anstie met with a case in which 3 grains (0.195 gm.) of morphin given as an injection, *per rectum,* caused death in sixteen hours."[49] On the other hand, the Lancet [50] records a case of a child only two years of age, who recovered after a dose of 1 grain (0.065 gm.) of the acetate. A number of similar cases, which are reported in medical journals and other literature, show that the susceptibility to the poisonous action of morphin is very great in some cases, and experience has taught the danger of giving a dose of this narcotic larger than one quarter of a grain, unless the medical attendant is aware of the peculiarity of his patient in this respect. It is obvious that too short an interval between the repetition of small doses may cause poisonous symptoms, as well as when the commencing dose is large. Dr. Taylor[51] shows that, in England, homeopathic physicians give doses of morphin—and without a written prescription—of size equal to those of physicians in regular practice, who write their prescriptions to be made up by the apothecary. He cites a case in his own experience, where a woman's life was imperiled in this way from alternate powders of morphin to the amount of ½ grain (0.032 gm.), and of sugar of milk without medicament. He suggests that, if the woman had died and left only one powder uneaten, it might have been found by analysis to contain only the innocent sugar of milk, and thus, upon the theory of the practice of a homeopathic practitioner, no suspicion would have attached to the poisonous character of the powder, because it would be supposed to contain only an infinitesimal amount of any medicine. We do not say that homeopathists in America practise in this way, but the writer has personal knowledge of a case occurring in his vicinity of a similar character to that quoted by Dr. Taylor. Dr. Eastes[52] has met with some homeopathic globules which contained 1 grain of morphin instead of $\frac{1}{1000}$ grain.

The fatal consequences arising from the constant and ignorant use of nostrums containing opium or its alkaloids, in domestic practice,

[48] Lancet, 1872, II. p. 24.
[49] *Ibid.*
[50] 1863, II. p. 8.
[51] Op. cit. p. 550.
[52] Lancet, Dec. 13, 1873.

are undoubtedly very frequent. There is a certain chronic poisoning induced by the use of this drug and its derivatives (especially morphin), which has very commonly been brought to notice. Paregoric and certain "soothing syrups and powders," given to children by ignorant persons in order to produce quiet and sleep, have been proved to be very injurious to health, and, in a few instances, death has been caused.[53] The children, however, become tolerant of the poison, and, though their death may not be immediately attributed to the continued use of the narcotic, yet their constitution becomes undermined. "In the late Mr. Granger's report on the Children's Employment Commission, it is stated that laudanum and other preparations of opium are given to young children in gradually increased doses, until the child will bear from 15 to 20 drops of laudanum at a time. The majority of these children die by the time they are two years old."[54]

The following is from the report of the superintendent of health and city registrar of Providence, Rhode Island, on the deaths in that city for March, 1873.[55]

"The decedent from poisoning, in March, was a child, killed by a dose of Mrs. Winslow's soothing syrup. It has long been known to physicians that the soothing properties of this popular medicine are due to opium in some form, and that the quantity of opium is so large as to make it a decidedly dangerous nostrum. There is no doubt that a considerable number of deaths every year should be recorded: 'Poisoned by Mrs. Winslow's soothing syrup.'"

The influence of idiosyncrasy in modifying the usual effects of opium is often seen, and may be of importance in legal medicine. Thus, Grisolle states that he saw narcotism induced in a lady by ½ grain (0.032 gm.) of opium. Dr. Christison mentions the case of a gentleman who was always narcotized by as little as 7 drops of laudanum, and Taylor observed alarming symptoms from the injection in a clyster of 1 grain of opium. Some diseases render the system extremely susceptible to its poisonous action, as has been observed in a previous chapter (§ 4), where this portion of our subject in relation to forensic medicine has been fully described. On the other hand, painful diseases enable the person to use very large doses, not only without injury, but with positive advantage; but it has been found by experience that it is a popular medical fallacy to suppose that morphin or

[53] California Medical Gazette, Nov. 1869; Am. Jour. of Pharm. May, 1872, p. 221, from Pacific Med. and Surg. Jour.

[54] Taylor, Med. Jurisprudence, 1865, p. 1154.

[55] Boston Med. and Surg. Jour. April, 1873, p. 432.

opium may be freely given so long as pain is experienced. There are many cases recorded where this fallacy has led to such free use of the anodyne that the patient suddenly becomes comatose and dies.

The habit of taking opium diminishes the influence of this drug upon the system, and doses, which in other cases would be absolutely poisonous, are taken with entire impunity by persons who indulge in this habit. When this habit has been acquired in later adult life, its effect upon the duration of life is not so well marked as in younger persons, yet recent knowledge shows that, as a general rule, opium eaters die from defects of interstitial nutrition or from cerebral diseases.[56] The picture of the opium eaters and smokers of the East, as drawn by travelers, is indeed a melancholy one, and their general testimony is that there it undoubtedly has the tendency to shorten life. This is the conclusion arrived at by Mr. Little, of Singapore, as the result of careful and extensive inquiries at that place of the owners of opium shops, of the smokers who frequented them, the prisoners in the house of correction, and the paupers at a poorhouse. The following picture of the effects of the habitual use of opium is drawn by this gentleman: "As the habit grows upon its unhappy victim, the first evils experienced are disturbed sleep, watchfulness, giddiness, sometimes headache, capricious appetite, a white tongue, frequently costiveness, indescribable oppression in the chest, and haziness of the eyes. Afterwards, a copious secretion of mucus takes place from the eyes and often from the nose also; digestion becomes much impaired, and micturition difficult; a mucous discharge begins to flow from the organs of generation; the sexual organs, at first preternaturally excitable, gradually lose their tone; the body wastes, the muscles lose their tonicity, and the bones are affected with dull, gnawing pains for some hours in the morning. By and by the figure stoops, and a peculiar shuffling gait is acquired, by which alone, a practised eye may recognize an old opium debauchee. At the same time, the eyebrow droops, the lower eyelid becomes dark, the eye itself seems to sink and grow dim, and the whole expression is that of premature old age. In both sexes the procreative power is greatly

[56] This subject received particular attention from Dr. Christison, in consequence of its importance in a remarkable civil trial. The Earl of Mar effected insurances on his life to a large amount, while addicted to the vice of opium eating, and died two years afterwards of dropsy. He had used laudanum for 30 years, at times to the amount of 2 or 3 ounces daily, and died at the age of 57. He suffered greatly from rheumatism. The insurance company, having been unaware of this habit, refused payment on the ground of its having a tendency to shorten life. The person holding the policy, therefore, instituted an action against the company, which was decided in favor of the former, but on other grounds.

lessened, and in those women who, nevertheless, do bear children, the secretion of milk is defective. The influence of the habit on the generative functions is indeed so decided that, were it not for fresh arrivals from China and other parts of the East, the population of Singapore would very soon be seriously diminished."[57] Finally, according to this author, structural derangement is induced, the digestive and assimilative functions become very much impaired, strumous affections are readily developed, and the opium smoker succumbs, without resistance, to any violent disease.

Some Eastern travelers, however, assert that the habit has no tendency to shorten life. Thus, Dr. Macpherson says that, although the habit of smoking opium is, in China, universal among rich and poor, we find them to be a powerful, muscular, and athletic people; and Dr. Burnes, who resided several years at the court of Scinde, says that "it will be found in general that the natives do not suffer much from the use of opium.[58] A celebrated Cutchee chief, who had taken opium largely all his life, was alive at the age of eighty, paralyzed by years, but his mind unimpaired."

Dr. Christison, moreover, found upon an examination of twenty-five cases, the particulars of which he obtained from various quarters, that instances of longevity among opium eaters in Great Britain were not uncommon. In most of these cases, it is expressly stated that no injurious effect upon the general health was observed; in some instances indeed, the persons being ruddy and robust in appearance. In a few, unpleasant symptoms were experienced only upon the intermission of the habit. The only inference that can at present be drawn from the testimony of travelers, and from these observations reported by Dr. Christison, is adverse to the general belief that opium, like intemperance in strong drink, has a tendency to shorten life when habitually used. Future observation must decide whether this reasonable belief is really the correct one. The possibility of abruptly discontinuing the habit without injury to the constitution has been shown by Dr. Christison.[59]

A more recent therapeutical authority on this subject of morphinism[60] writes in regard to this subject as follows:—

"A statement of the extreme amounts of morphin taken by persons habituated to its use would have but little value, and could perhaps be paralleled from the experience of a large proportion of the readers

[57] Edin. Month. Jour. June, 1850.
[58] Christison on Poisons.
[59] Edin. Month. Jour. June, 1850.
[60] Therapeutic Handbook of the United States Pharmacopœia, Robt. T. Edes, M. D. of New York, 1883.

of this book. A somewhat extended inquiry among druggists in Massachusetts, by Dr. F. E. Oliver, revealed the fact that 1 dram (4 gm.) per week is by no means an uncommon allowance for a regular consumer. The dangers of a confirmed morphin habit have probably been somewhat exaggerated, so far as the bodily health is concerned. There is no doubt that a person may be an habitual user of morphin for years, and retain a reasonable degree of health, sustaining himself nearly as usual in society and business, and keeping his stimulus from being suspected. The effects most obvious to himself will be digestive disturbances in the form of loss of appetite, dyspepsia, constipation, and emaciation; and, on the part of the nervous system, loss of mental energy, clearness, and decision, and extreme and indescribable distress, as the effect of each dose passes off, and lasting until its repetition. The most good and least bad effects may be attained by always using the smallest possible dose which will give the desired effect, and increasing as slowly as possible. The usual tendency among habitual users of morphin for purposes of stimulation is to increase the dose rapidly. The habit is difficult, but by no means impossible, to escape from. Unfortunately it is far from rare, and it is practised in both ways, the ordinary method by the mouth and the subcutaneous injection, the latter causing apparently less disturbance of the digestion. The physician should never put a hypodermic syringe into the hands of a patient for habitual use, except under extreme necessity; and if obliged to do so occasionally, should withhold all instructions which would enable the patient to continue the administration without medical advice. Many so-called opium antidotes are solutions of morphin."

There is no doubt in the minds of medical men of the present time that the habit of opium eating is injurious to health, and therefore calculated to shorten life. In any proposal for life insurance, the insurers ought to be informed of this habit, where it exists, and no medical man should sanction its concealment merely because many persons addicted to the habit have lived for years in tolerable health. The practice often is, and may be, concealed from a medical attendant; then the insured, if not candid in avowing its existence, must expose his representatives to the risk of losing all benefit under the policy.[61]

764. Poisoning by morphin or its salts.—Poisoning by morphin or its salts requires no further consideration than has been given above,

[61] Taylor, Med. Jurisprudence, 1865, p. 1155.

since opium owes its poisonous nature principally to this alkaloid, and the intensity of the effects of poisoning by any preparation of opium is directly proportionate to the amount of morphin present in the opium. The symptoms are rather more prompt in their appearance, but are otherwise entirely similar to those produced by opium or laudanum. For an adult male, the medicinal dose of morphin, its acetate, hydrochlorate, or sulphate, is 1/6 grain (0.011 gm.). Death has been caused by 1 grain (0.065 gm.) of the hydrochlorate taken in divided doses over a period of six hours,[62] and by 1⅓ grains (0.087 gm.) within four hours.[63] The former dose was equal, probably, to 6 grains of opium. For an adult female the medicinal dose is ⅛ grain (0.008 gm.). For a child the proportionate dose should not be more than that fraction of the dose prescribed for adults, found by dividing the child's age, in years completed, by the number twenty-four; *viz.*, for a female child under one year, 1/96 grain (0.0006 gm.) of morphin. It was for poisoning with the acetate of morphin that Castaign who had formerly been a pupil of Orfila, was, in the year 1823, tried and executed in Paris. He was convicted less upon the medical than the circumstantial evidence offered, since with the most ingenious refinement of cruelty, he had administered tartar emetic to his victim for the purpose of getting rid of any of the poison which might have remained in the stomach. The medical testimony could only show that the symptoms and post-mortem appearances were not opposed to the supposition that morphin was the cause of death.

765. **Compound poisoning with opium or morphin.**—It would be impossible to describe in this treatise the symptoms of compound poisoning in such a manner as to prevent a confused idea. A medical expert should be competent, from a knowledge of the symptoms presented in any given case, to explain the modifications of opium poisoning by the control of another poison (see § 46), from a knowledge acquired of the symptoms caused when either poison was administered alone; but the predominance of these symptoms will depend upon the more active of the two or three drugs used together, or the interval of time elapsing between the period at which each was administered, or the relative size of the dose taken. After the general remarks upon this subject in our opening chapter further comment is unnecessary.

766. **Treatment.**—In acute cases, the same treatment as with alcohol; empty the stomach, and arouse the patient to respiratory

[62] Ed. Month. Jour. Sept. 1846. [63] *Vide*, § 763.

efforts; flip the surface of the body with the end of a wet towel or with fine birches; this flagellation will excite the capillary circulation and promote oxidation. In a case treated by the writer, where the respiration of an adult was reduced to four feeble movements of the chest, this method of treatment, carefully performed, improved his condition each five or ten minutes, so that in the course of half an hour the patient could be lifted on his feet, and he could walk around the room, three persons holding him erect, one of whom forced locomotion by pushing the legs from behind; in the course of an hour the patient made voluntary movements of locomotion and was gradually restored to consciousness. This illustration is offered simply to show that persistency, even with this simple means of resuscitation, may be expected to produce a successful result. Artificial respiration, if practised, should be at the rate of eighteen times a minute, and should be resorted to in case the efforts to breathe on the part of the patient are not noticeable. The state of the pulse rate is not of much consequence, as the respiratory movements are the first and most important to be obtained. Recently, a treatment by the administration of permanganate of potash has been advocated for the purpose of accomplishing the oxidation of morphin in the tissues. This remedy is recommended in doses of 1 grain (0.06 gm.) more than the quantity of morphin which has been taken, the remedy being dissolved in 30 or 40 parts of water; this should be taken by the mouth. The use of atropin as a counter-poison against opium and morphin is still a subject of contention among the best authorities. It is true, however, that atropin is a valuable stimulant to excite respiratory movement, and, if used for this purpose only, would, undoubtedly, in some cases, be very efficient. There is a case, mentioned by Cruse,[64] of an infant, a week old, who was accidentally poisoned by a grain of morphin. Nothing would relieve the coma resulting from this dose, though various treatments were tried for several hours, until ¼ grain (0.016 gm.) of atropin was administered, this dose being repeated at the interval of half an hour; recovery was complete in thirty-six hours with no untoward symptoms resulting from the administration of such a large dose of atropin. Atropin, however, may prove a dangerous remedy if administered too late; the usual dose for subcutaneous injection, when the period is early after that of poisoning, would be from 1/60 to 1/20 of a grain (0.001 to 0.003 gm.). Its repetition should be followed cautiously by referring to the dilatation of the pupils as representing the absorption of atropin.

[64] Archiv. f. Kinderheilk. 1893.

Prof. H. C. Wood advises strongly the use of strychnin in doses of 1/32 of a grain (0.002 gm.) subcutaneously introduced in order to excite the respiratory movements. The use of strong coffee is also advised; it is beneficial as a common remedy to stimulate the heart and lung nerve centers. Cocain hydrochlorate is also indicated in many cases to be administered subcutaneously in doses of from ¼ to ½ of a grain (0.016 to 0.032 gm.). Reference to the previous article on cocain (see *supra,* § 689) will show the reader that large quantities of cocain can be administered to persons in the habit of taking morphin, without serious dangerous symptoms; so that, in the case of acute poisoning from morphin, larger doses would apparently be indicated to combat by cocain absorption the poisonous action of morphin, without ascribing the explanation of this apparent tolerance of cocain to morphinism. It is suggested that the action of morphin in preventing oxidation of the tissues may retard the absorption of cocain.

767. **Action.**—The action of morphin on the central nervous system is, first, that of an excitant, which is then followed by depression. The depressing action upon the brain mass—especially of the higher intellectual faculties—seems to be produced very soon after the first excitation has appeared, while the stimulating action upon the spinal cord persists for a longer period. In consequence of this, the medulla oblongata, which is at the summit of the spinal cord, seems to show its depressing action,—or, in other words, an inhibition of its functions,—especially in retarding the movements of respiration. On the other hand, a depressing action upon the brain mass appears to render the patient unconscious of an existing pain, which, carried to a higher extent, will produce sleep and unconsciousness. The reflex action of the periphery of the nervous system, whose action is at the spinal cord, may persist even after the period of unconsciousness, that is to say, the irritation of a noise will cause the patient to start; pinching of the skin will start a quick reflex response; and persistent tactile irritation of the surface of the skin, as by two pointed instruments separated in their contact, can be discerned as easily by the patient as when not under the influence of morphin. We have mentioned as one of the effects from the narcosis of morphin poisoning the "Cheyne-Stokes" form of respiration. This has been more particularly described in detail by Loewy who found that larger quantities of carbon dioxid in the tissues were needed to increase the volume of air received into the lungs. This might explain the peculiar slowing action developed, because opium retards oxidation of the

tissues, and the blood carried to the brain would contain less carbon dioxid than in health.

Morphin has but little action on the central organ of circulation, except from the secondary action of the failure of the oxidation process by the retarded respiratory movements. The blood pressure remains at a high tension, and the capillary and peripheral arteries show no change of lumen; the consequence of this is dilatation of the veins, suffusion of the countenance, and cyanosis of the skin; the face having, sometimes, a bloated look. Exciting contraction of the superficial capillaries brings warmth to the surface, and encourages respiration. It should be specially remarked that the main action of morphin is exerted upon the respiratory, rather than upon the cardiac, movements.

The action of opium upon dogs is different from that on man; the respiration being more rapid and stertorous, and the reflex actions less marked. There is a partial paralysis of the hind legs, which gives a "hyena"-like attitude to the dog; convulsions are more common in them. Goats will take larger doses in proportion to their size than would be supposed. A young goat weighing 30 kilos could take 195 grams and recover; it would seem that the lethal dose is estimated at 0.25 to 0.30 gm. per kilo of the goat's weight.[64a] The action upon cats is a remarkable degree of excitement, increasing in proportion to the dose given, producing convulsions and death, without the intervention of sleep. Pigeons are specially immune from reasonable doses of opium mixed with their food. One pigeon is said to have consumed 801 grains of opium in its food during a period of fourteen days after receiving the poison. The action on man is sometimes very rapid, as related by Tardieu; in other cases, which are not common, there may be no coma, but convulsions. The sudden action, mentioned by Tardieu, is that in which a person sinks into a deep sleep within five of ten minutes, and dies in a few hours.

768. Diagnosis.—The diagnosis of opium poisoning may be confused with poisoning by some other narcotic substances, as, for instance, alcohol, chloral, belladonna, and carbon monoxid gas; but the absence of the smell of either of these substances will assist in distinguishing between them. The difference between the convulsions of opium and strychnin is sufficiently evident not to confuse these two poisons.

769. Post-mortem appearances.—The post-mortem appearances show

[64a] Guinard, in Compt. Rend. CXVI. pp. 520–522.

hyperemia of the brain, sometimes with serous effusions, and moisture upon the surface of the membranes.

It is quite unnecessary to particularize the morbid alterations which have been seen in persons dying from poisonous doses of opium, since there are none which are sufficiently apparent or distinctive to be attributed to this cause. As a general rule, the vessels of the brain and spinal marrow will be found turgid, and the lungs and other vascular organs congested, while the blood remains fluid. The multitude of diseases and of modes of death by asphyxia which may occasion these conditions precludes us from attaching much importance to them as indicative of death from opium. Sometimes opium in substance or laudanum may be found in the stomach, or the latter may be recognized in this organ by its smell; but in the vast majority of cases the poison is eliminated from the system, so that at the post-mortem inspection no trace of it will be found except in urine.

Though the lesions which have been noticed in the organs of those poisoned by opium have no very specific character, still they are almost always constant. The stomach and intestines are sometimes colored on their interior aspect by the saffron-yellow tint of opium. The mucous membrane shows varied and striking tints, formed by the mixture of the coloring matter of the medicament with the arboriform injections of the blood vessels, thus adding a vivid red to the yellow color. (Tourdes.)

M. Tardieu, in his excellent treatise on Poisons,[65] quotes the detailed account of a post-mortem examination on the body of an old man, poisoned by about an ounce of laudanum, as given by Tourdes:[66] The autopsy was made sixty-two hours after death. Rigor mortis was general and well pronounced; there was no sign of putrefaction; the abdomen had no bluish discoloration, though somewhat tympanitic. There was nothing remarkable about the expression of the face; the face was not discolored; the eyelids had a bluish tinge. The pupil was moderately dilated, its diameter being 4 millimeters. The dura was somewhat injected. The whole surface of the body was pale. There was no sign of papular eruption; but the skin of the chest and neck presented in an unmistakable degree the condition of papillary elevations known as "gooseflesh." There were several spots of laudanum on both hands. The skin of the scrotum was strongly injected; there was no erection of the penis; at the meatus urinarius a whitish, milky fluid was found, which, on micro-

[65] Op. cit. 2d ed. p. 1052.
[66] Gazette Médicale de Strasbourg, 1858, p. 102.

scopical examination, was seen to contain spermatozoa. The tongue, which lay behind the dental arch, was yellowish in color, and slightly injected at the base. The soft palate and the pharynx were of a reddish tinge. The upper part of the œsophagus was of a pale pink color; the lower portion was bright red.

The stomach was remarkable on account of the yellowish, greenish, and reddish appearance of its mucous membrane; the yellowish, greenish, and saffron color predominated in the right half; a strong reddish injection was particularly noticeable in the left half. Some red spots with a tinge of purple were observed in the yellowish discoloration extending over a great part of the organ. These spots were formed either by bright, very limited injections, or by bloody effusions. The mucous membrane was of the usual consistence, and showed no trace of erosion or ulceration. The stomach contained but a small amount of yellowish matter colored, evidently, by laudanum. There was a sour smell. The duodenum presented the same yellowish discoloration with red spots and arborizations; its mucous membrane was intact. It contained some yellowish matter like that found in the stomach, but in greater abundance. The yellowish discoloration extended into the small intestine to a point 2 meters distant from the pylorus; beyond this point the mucous membrane was of normal appearance. The liver was normal in color and size. There was a small amount of bile in the gall bladder. The spleen weighed 410 grams; its external covering was enveloped with old false membranes of a thickness of 1 to 2 centimeters. The right kidney was enormous; it contained a calculus as large as an elongated hazelnut, filling its pelvis, and taking its shape. The calculus was formed of uric acid and urate of ammonium.

The bladder contained a small amount of urine having an acid reaction. The pericardium contained a small quantity of serous fluid of alkaline reaction. The heart was very large, weighing 620 grams; the left ventricle was much hypertrophied, the thickness of the walls varying from 2½ to 3½ centimeters. The arch of the aorta was dilated. The heart contained some fibrinous, dense, colorless clots which had prolonged themselves like polypi into the aorta and pulmonary artery. A small amount of reddish and grumous blood flowed from the pulmonary veins. Blood, which was more fluid, flowed from the superior vena cava. The lungs were rose-colored and crepitant. There was a well-marked pulmonary congestion. The dura mater was much congested. The subarachnoid fluid was extremely abundant, and gave the brain the appearance of a gelatiniform mass; the reaction of the fluid was alkaline. The arach-

noid presented some old milky spots. The vessels of the pia mater were much injected. The cerebral ventricles contained a small amount of serous fluid. The cerebral parenchyma was markedly injected, which injection was equally pronounced at the front and the back. The chemical analysis performed by M. Hepp, chief pharmacist of the civil hospital, demonstrated the presence of morphin in the substance extracted from the stomach and duodenum.

At first sight it would seem reasonable to attribute the post-mortem effects of morphin to poisoning by nitric acid, were it not that the absence of injury to the tissue removes the doubt. Generally, the mucous membrane of the alimentary canal is free from alteration. The surface of the body is completely discolored, and animal heat persists for a long time, even after the invasion of cadaveric rigidity.

The following case of the autopsy of a suicide by morphin is reported by Medical Examiner Draper: (Case 220, 1894), aged fifty years. Chronic nephritis. Heart normal. Spleen, pancreas, stomach, intestines, liver, and bladder normal. Kidneys dense and opaque; on section, obscure outlines of elements. Brain, vessels of pia mater and brain substance injected; many punctate hemorrhages in corpus striatum.

770. **Meconic acid.**—The characteristics of this acid are as follows: It reddens the neutral sesquisalts of iron; the red color is destroyed by alkalies, protochlorid of tin, and nitric acid, assisted by heat. This, which is the most reliable, test for meconic acid, is still open to objection. Thus, sulphocyanic acid produces a similar red color with the persalts of iron. The force of this objection is derived from the fact that sulphocyanic acid is naturally sometimes present in the saliva. Christison, indeed, states that "it is seldom possible to procure a distinct blood-red coloration from the saliva, except by evaporating a large quantity to dryness, and redissolving the residue in a small quantity of water;" but Pereira dissents from this statement, and says that in a large majority of cases he has found saliva distinctly and unequivocally reddened by the persalts of iron. He says, moreover, that he has several times obtained from the stomach of subjects in the dissecting room a liquor which reddens the salts of iron. We believe that the opinion of chemists now is, in general, in accordance with the statements of the last-named author. The means of distinguishing the sulphocyanid from the meconate are to be found in the action of chlorid of gold, or of corrosive sublimate; since, if a few drops of a solution of either of these reagents be added to the red liquid, the color, if due to sulphocyanic acid, will be immediately de-

stroyed. Further, the liquid may be diluted and a few drops of a solution of the acetate of lead added; a precipitate will fall, which is either meconate or sulphocyanid of lead. The former is insoluble, while the latter is quite soluble, in acetic acid.

Meconic acid is also precipitated from its solutions by chlorid of calcium or chlorid of barium, into the form of crystals of meconate of calcium or barium. The acetate of lead is the best reagent to use in separating meconic acid from organic mixtures, since the precipitated meconate of lead can be collected upon a filter paper, suspended in water, and decomposed by sulphureted hydrogen, meconic acid remaining in solution in the water, and the black sulphid of lead being precipitated; this last can be separated by filtration, and the excess of sulphureted hydrogen expelled from the filtrate by warming, when we have left a tolerably pure solution of meconic acid which can be tested as above mentioned.

771. Narcotin.—This alkaloid usually crystallizes in the form of rhombic prisms. The crystals are soluble in 25,000 parts of cold, and in 7,000 parts of boiling, water; but when amorphous, as when separated from its salts by ammonia water, it is soluble in 1,500 parts of cold, and in 600 parts of boiling, water. It is soluble in 120 parts of cold, and in 20 to 24 parts of boiling, alcohol; in 126 parts of cold, and in 48 parts of boiling, ether; it is very soluble in chloroform (2.69 parts), in 60 parts of acetic ether, and in about 22 parts of benzol.[67] It is quite soluble in dilute acids, except acetic, and is easily precipitated from these solutions by ammonia water in crystalline form. The best test for narcotin is that recommended by Husemann. This consists in treating a little narcotin (one half of a milligram is sufficient) with 0.2 of a cubic centimeter of concentrated sulphuric acid; or, dissolve it in dilute sulphuric acid (1 : 5) and evaporate this solution slowly, when a red residue will be left, which, when touched with a trace of nitric acid, becomes violet. If the sulphuric acid residue is heated to 200° C. (392° F.), it becomes deep violet-red in color; and the fact that this color is not produced below 100° C. (212° F.) enables us to distinguish narcotin from curarin, which latter gives the same color with sulphuric acid at 100° C. Fröhde's reagent gives a green color with narcotin.

772. Detection of opium or one or more of its constitutents.—The recognition of opium depends upon the isolation and detection of one or more of its constituents, the two of especial importance being me-

[67] Dragendorff, loc. cit.

conic acid and morphin, since they exist in opium to a larger extent than any of the other constituents; it is probable, however, that the detection of narcotin, which is present in opium to the extent of 6 or 8 per cent, will in the near future constitute an important factor in the chemistry of opium poisoning. The narcotin is, however, mostly removed from some of the pharmaceutical preparations of opium, such as the "denarcotized opium" (U. S. P., 1890), and the "deodorized tincture of opium" (U. S. P., 1890). If, in a chemico-legal analysis we find both meconic acid and morphin in the contents of the stomach or vomitus, it shows that the form of the poison taken was opium itself or one of its preparations, whereas if morphin alone is present without meconic acid, the form of poison used must have been morphin or one of its salts. If a notable amount of narcotin were detected, it would prove that the form of poison used was one of the preparations of opium from which the narcotin had not been removed, such as opium itself and laudanum.

773. Separation of opium or its constituents from organic mixtures.—Various processes have been recommended for this purpose. The principal ones are those recommended by Stas and by Dragendorff. We give them both in some detail, since they are applicable to the isolation of very many of the vegetable and organic poisons, as well as that of opium and morphin.

The method of Professor Stas, of Brussels, justly distinguished for his admirable reports in the Bocarmé case, is detailed by him as follows: "The method I now propose for detecting the alkaloids in suspected matters, is nearly the same as that employed for extracting those bodies from the vegetables which contain them. The only difference consists in the manner of setting them free, and of presenting them to the action of solvents. We know that the alkaloids form acid salts, which are equally soluble in water and alcohol; we know also that a solution of these acid salts can be decomposed, so that the base set at liberty remains either momentarily or permanently in solution in the liquid. I have observed that all the solid and fixed alkaloids above enumerated, when maintained in a free state and in solution, in a liquid, can be taken up by ether when this solvent is in sufficient quantity. Thus, to extract an alkaloid from a suspected substance, the only problem to resolve consists in separating, by the aid of simple means, the foreign matters, and then to find a base, which, in rendering the alkaloid free, retains it in solution, in order that the ether may extract it from the liquid. Successive treatment by water and alcohol, of different degrees of concentration, suffices

for separating the foreign matters, and obtaining in a small bulk a solution in which the alkaloid can be found. The bicarbonates of potash or soda, or these alkalies in a caustic state, are convenient bases for setting the alkaloids at liberty, at the same time keeping them wholly in solution,—especially if the alkaloids have been combined with an excess of tartaric or of oxalic acid." To put in practice the principles thus explained, the following method is proposed: "I suppose that we wish to look for an alkaloid in the contents of the stomach or intestines; we commence by adding to these matters twice their weight of pure and very strong alcohol; we add, afterwards, according to the quantity and nature of the suspected matter, from 30 to 45 grains (2 to 3 gm.) of tartaric or oxalic acid—in preference, tartaric; we introduce the mixture into a flask, and heat it to 160° or 170°F. After it has completely cooled, it is to be filtered, the insoluble residue washed with strong alcohol, and strained, and the filtered liquid evaporated *in vacuo,* or it may be exposed to a strong current of air at a temperature of not more than 90° Fahrenheit. If, after the volatilization of the alcohol, the residue contains fatty or other insoluble matters, the liquid is to be filtered a second time, and then the filtrate and washings of the filter evaporated in the air pump till nearly dry. If we have no air pump, it is to be placed under a bell jar, over a vessel containing concentrated sulphuric acid or quicklime. We are then to treat the residue with cold anhydrous alcohol, taking care to exhaust the substance thoroughly; we evaporate the alcohol at a low temperature, or better still *in vacuo,* spontaneously. We now dissolve the acid residue in the smallest possible quantity of water, introduce the solution into a small test tube, and add, little by little, pure powdered bicarbonate of soda or potash, till a fresh quantity produces no further effervescence of carbonic acid. We then agitate the whole with four or five times its bulk of pure ether, and leave it to settle. When the ether swimming on the top is perfectly clear, then decant carefully about two cubic centimeters of it into a small glass capsule, and leave it in a very dry place to spontaneous evaporation." If the suspected alkaloid is solid and fixed, there may or may not be a residue containing it. If there is, a solution of caustic potash or soda should be added to the liquid, and agitated briskly with ether. "This dissolves the vegetable alkaloid, now free, and remaining in the solution of potash or soda. In either case we exhaust the matter with ether. Whatever be the agent which has set the alkaloid free,—whether it be the bicarbonate of soda or potash, or caustic soda or potash,—it remains, by the

evaporation of the ether on the side of the capsule, as a solid body, but, more commonly, a colorless milky liquid, holding solid matters in suspension. The odor of the substance is animal, disagreeable, but not pungent. It turns litmus paper permanently blue."

In order now to obtain the solid alkaloid in a crystalline state, the foreign matters, with which it is generally associated, must be first removed. Prof. Stas, to accomplish this purpose, adds a few drops of water, feebly acidulated with sulphuric acid, to the contents of the capsule, and thus forms an acid sulphate, which should be carefully decanted, evaporated *in vacuo* or over sulphuric acid, the residue treated with pure carbonate of potassium, and the alkaloid dissolved out by absolute alcohol. The evaporation of the alcohol gives the alkaloid in crystals. By this process, Prof. Stas has isolated all the important fixed alkaloids previously mixed with foreign matters.[68]

The following is the detailed process of Dragendorff,[69] which the writer much prefers to any other method for the isolation of organic principles, poisonous or otherwise, from ordinary organic mixtures, such as animal tissues and fluids, articles of food, etc. The chemical principles upon which the process depends are the same as in Stas's process. The detailed scheme is given below, in order to avoid the necessity of repetition in speaking of the organic poisons to be considered hereafter:—

I. Extract with water acidulated with sulphuric acid two or three times at 40°–50° C. for several hours. Strain and filter the united extracts.

II. Evaporate to beginning syrupy consistence, mix the residue with three or four times its volume of alcohol, macerate twenty-four hours at about 30° C., cool, and filter. Wash the solid upon the filter with 70 per cent alcohol.

III. Evaporate off the alcohol, transfer the residue to a flask, cool, and, if necessary, dilute and filter. Shake this aqueous acid fluid with freshly rectified naphtha ("petroleum ether") at the ordinary temperature, frequently and vigorously. After the two fluids have separated, decant the naphtha and allow it to evaporate in several watch glasses. The shaking with naphtha should be repeated as long as anything is removed by it. The naphtha removes

[68] Am. Jour. of Pharmacy, Jan. 1853.
[69] Ermittelung von Giften, 1876, p. 141.

from the fluid certain impurities (coloring matters, etc.) in addition to the following substances:

NAPHTHA RESIDUE FROM THE ACID FLUID.

1. *Crystalline.*

a. Yellowish and volatilized with difficulty.

(*a*) The crystals dissolve in concentrated sulphuric acid with a pale yellow and, later, brown and greenish-brown color. **Piperin.**

(*b*) The solution in sulphuric acid remains yellow. Potassium cyanid and potassium hydrate color it blood-red on warming. **Picric acid.**

b. Colorless, easily volatile, and with a strong odor. **Camphor and similar substances.**

2. *Amorphous.*

a. Nonvolatile.

(*a*) Concentrated sulphuric acid dissolves it immediately with a violet and, later, a greenish-blue color. **Constituent of the black hellebore.**

(*b*) Concentrated sulphuric acid dissolves it with a yellow color, which gradually changes to a violet-red and then to a roe-brown. **Constituent of the aconite and a decomposition product of aconitin.**

b. Pale, with a sharp taste, and reddens the skin. **Capsicin.**

3. *Fluid and with a strong odor.* **Ethereal oils, carbolic acid, etc.**

IV. The aqueous fluid is next shaken in the same way with benzol. The benzol is decanted into several watch glasses and allowed to evaporate.

BENZOL RESIDUE FROM THE ACID FLUID.

1. *Crystalline.*

a. Well-formed colorless crystals.

(*a*) Sulphuric acid dissolves the hair-like crystals colorless. Evaporation with chlorin water leaves a residue which gives the murexid test with ammonia. **Caffein.**

(*b*) Sulphuric acid dissolves the rhombic crystals colorless. Mixed with oil and applied to the skin the substance blisters. **Cantharidin.**

(*c*) Sulphuric acid dissolves the scaly crystals, at first colorless, slowly changing to red. It does not blister. Warm alcoholic potassium hydrate gives a temporary red color. **Santonin.**

(*d*) Sulphuric acid dissolves the racemose crystals with a yel-

lowish-orange color, and this solution gives a transient violet color with nitric acid. **Caryophyllin.**

(*e*) Sulphuric acid colors the crystals nearly black, and becomes itself colored a beautiful red. **Cubebin.**

b. Crystals pale or clear yellow. (*a*) **Piperin** (III. 1, a, *a*) (*b*) **Picric Acid** (III. 1, a, *b*).

(*c*) Potassium hydrate = purple color. **Aloetin.**

c. Usually only indistinct colorless crystals.

(*a*) Sulphuric acid = greenish-brown. Bromin colors this solution red, which becomes green again on diluting with water. The substance shows the action of a frog's heart. **Digitalin.**

2. *Amorphous.*

a. Colorless or pale yellow residue.

(*a*) Sulphuric acid gives, at first, a yellow solution, which becomes red later. Fröhde's reagent does not give a violet color. **Elaterin.**

(*b*) Sulphuric acid gives a red solution. Fröhde's reagent a violet-red. Tannin gives no precipitate. **Populin.**

(*c*) Sulphuric acid gives a deep red color. Fröhde's reagent = beautiful cherry-red. Tannin=yellowish-white precipitate. **Colocynthin.**

(*d*) Sulphuric acid = a beautiful red, often gradual. Tannin = no precipitate. **Constituents of the Pimento.**

b. Clear yellow residue.

(*a*) Sulphuric acid = yellow. This solution becomes green, then quickly blue and violet, upon the addition of nitric acid. **Colchicin.**

(*b*) Sulphuric acid dissolves it with the separation of the violet powder. Potassium hydrate=red. Sulphid of ammonium=violet, and, on heating, indigo blue. **Chrysammic Acid.**

c. Greenish bitter residue. Sulphuric acid = brown. Fröhde's reagent=at first brown, then green, blue-violet, and finally violet, the change of color beginning at the edge. **Constituents of the wormwood, absinthin.**

V. The aqueous acid fluid is now shaken with chloroform, which is removed with a pipette and allowed to evaporate in watch glasses.

CHLOROFORM RESIDUE FROM THE ACID FLUID.

1. *More or less distinctly crystalline.*

a. The solution of the sulphate reacts like an alkaloid with a solution of iodin in iodid of potassium.

(*a*) Sulphuric acid = colorless solution. Chlorin and ammonia water do not give murexid reaction. **Cinchonin.**

(*b*) Sulphuric acid = colorless solution. Chlorin and ammonia water give the murexid test like caffein. **Theobromin.**

(*c*) Sulphuric acid = colorless solution in the cold, but on warming=blue-violet. **Papaverin.**

(*d*) Sulphuric acid=blue in the cold. **Unknown impurities in commercial papaverin.**

(*e*) Sulphuric acid=at first gray-brown and in about twenty-four hours blood-red. Iodin water=blue. **Narcein.**

b. It does not react like an alkaloid.

(*a*) Sulphuric acid=beautiful yellow. Mixed with niter, then moistened with sulphuric acid, and finally treated with concentrated sodium hydrate, it assumes a brick-red color. **Picrotoxin.**

(*b*) Sulphuric acid=beautiful red. It produces slowing of a frog's heart. **Helleborein.**

2. *Amorphous.*

a. The solution in acetic acid produces slowing of a frog's heart, or local anesthesia.

aa. Does not produce local anesthesia.

(*a*) Sulphuric acid=reddish brown. Bromin colors this solution beautiful purple, which changes again to green on diluting. Hydrochloric acid=greenish-brown. **Digitalein.**

bb. Produces local anesthesia.

(*a*) Sulphuric acid=brown. This solution becomes violet with the absorption of water. **Saponin.**

cc. Sulphuric acid=dirty red. Hydrochloric acid=red-brown in the cold, but on boiling, brown. **Constituents of the hellebore, principally jervin.**

VI. The acid fluid is again shaken with naphtha in order to remove the last traces of chloroform. The naphtha is removed and the fluid is made alkaline with ammonic hydrate.

VII. Shake the ammoniacal fluid with naphtha at the ordinary temperature.

After the separation of the two fluids decant a part of the naphtha into two watch glasses, one of which has been moistened with concentrated hydrochloric acid.

NAPHTHA RESIDUE FROM THE AMMONIACAL FLUID.

1. *Crystalline.*

a. Crystals volatilize with difficulty.

aa. Sulphuric acid=colorless.

(*a*) Potassium bichromate=blue, then red, in sulphuric acid solution. **Strychnin.**

(*b*) Potassium bichromate does not give blue color. Chlorin water and ammonia water give thalleioquin reaction. **Quinin.**

b. Sulphuric acid = yellow, gradually changing to a beautiful deep-red. **Sabadillin.**

c. The crystals are easily volatile. **Conhydrin.**

2. *Amorphous.*

(*a*) Purest sulphuric acid=almost colorless solution. Sulphuric acid which contains nitric acid=red color which changes to orange. **Brucin.**

(*b*) Sulphuric acid=yellow color which changes to a deep red. **Veratrin.**

(*c*) Sulphuric acid=brown-green. Fröhde's reagent=red color which soon becomes green. **Emetin.**

3. *Volatile and with a strong odor.*

a. Crystalline residue on watch glass moistened with hydrochloric acid.

aa. Bichlorid of platinum=no precipitate in its solution.

(*a*) The crystals of the hydrochloric acid compound act upon polarized light, and are mostly needle-shaped and prismatic. **Coniin and methylconiin.**

(*b*) Crystals are cubes or tetrahedra. **Alkaloid of the Capsicum.**

b. The hydrochloric acid compound is amorphous, or only crystalline after decomposition.

aa. Bichlorid of platinum=precipitate in its dilute aqueous solution.

(*a*) The hydrochloric acid salt treated as quickly as possible with Fröhde's reagent gives in about two minutes, a deep violet solution which gradually fades. **Lobelin.**

(*b*) The hydrochloric acid salt smells like nicotin. Fröhde's reagent = yellowish color, which becomes pale red after twenty-four hours. **Nicotin.**

bb. Bichlorid of platinum=no precipitate in its dilute solution.

(*a*) Its naphtha solution gives no turbidity with a solution of picric acid in naphtha, but the mixture leaves a crystalline residue after evaporation (mostly triangular plates.) **Trimethylamin.**

(*b*) Its naphtha solution treated in the same way gives moss-like crystals. It is colored blue by a solution of calcium hydrochlorite, and also by dilute sulphuric acid and potassium bichromate. **Anilin.**

VIII. Shake the ammoniacal fluid with benzol.

BENZOL RESIDUE FROM THE AMMONIACAL FLUID.

1. *Mostly crystalline.*

a. Sulphuric acid=colorless solution which does not become colored either by standing or by the addition of nitric acid.

aa. Dilates the pupil of a cat's eye.

(*a*) Bichlorid of platinum=no precipitate in aqueous solution. The sulphuric acid solution has a peculiar odor on warming. **Atropin.**

(*b*) Bichlorid of platinum=precipitate when used in exactly the right amount. **Hyoscyamin.**

bb. Does not dilate the pupil.

(*a*) Sulphuric acid solution colored blue by potassium bichromate.

(*aa*) Tetanizes a frog. **Strychnin.**

(*bb*) Slows the respiration in a frog. **Ethyl- and Methyl-strychnin.**

(*b*) Sulphuric acid and potassium bichromate do not give blue color.

(*aa*) The dilute sulphuric acid solution fluoresces and gives the thalleioquin reaction. **Quinin.**

(*bb*) The solution does not fluoresce. **Cinchonin.**

b. Sulphuric acid=at first colorless solution, which becomes, on standing, rose-red or bluish-violet, and blood-red or brownish on the addition of nitric acid.

(*a*) A solution of dilute sulphuric acid, on heating, gradually becomes deep blood-red, violet when cooled and treated with nitric acid. The dilute sulphuric acid solution gives a precipitate with ammonia water. **Narcotin.**

(*b*) The dilute sulphuric acid solution usually becomes blue on heating. An excess of ammonia water = no precipitate in dilute solutions. **Codein.**

c. Sulphuric acid=yellow.

(*a*) The solution remains yellow on standing. **Acolyctin.**

(*b*) It becomes beautiful red on standing. **Sabadillin.**

d. Sulphuric acid = deep reddish-brown immediately. **Thebain.**

2. *Mostly amorphous.*

a. Purest sulphuric acid=colorless or pale-red or yellow.

(*a*) The sulphuric acid solution is immediately colored red by nitric acid, then orange. **Brucin.**

(*b*) The solution gradually becomes brownish-red. Calcium hypochlorite colors it red. It contracts the pupil. **Physostigmin.**

b. Pure sulphuric acid=yellow solution, which later becomes red. (In the case of delphinin more quickly and more of a dark cherry-red.)

(*a*) The hydrochloric acid solution becomes red on heating.

(*aa*) It causes retching in frogs, and in large doses tetanus. Veratrin.

(*bb*) Without action on frogs. **Sabatrin.**

(*b*) The hydrochloric acid solution does not become red. **Delphinin.**

c. Pure sulphuric acid=yellow, then reddish-brown and gradually violet-red.

(*a*) In small doses paralyzes frogs. Dilates pupil of cat's eye. Difficultly soluble in ether. **Nepaline.**

(*b*) Much more feeble physiological action. It usually does not dilate the pupil. Very soluble in ether. **Aconitin.**

(*c*) Very feeble physiological action. Does not dilate pupil. Difficultly soluble in ether. **Napellin.**

d. Sulphuric acid = dark grayish-brown, changing in a few seconds to blood-red. **Alkaloid in the Aconitum lycoctonum.**

e. Sulphuric acid=brown-green. Fröhde's reagent=red changing quickly to a green. **Emetin.**

IX. Shake the ammoniacal solution with chloroform.

CHLOROFORM RESIDUE FROM THE AMMONIACAL FLUID.

a. Sulphuric acid dissolves it colorless in the cold.

aa. It becomes slightly colored on warming.

(*a*) After cooling, nitric acid=blue-violet. Sesquichlorid of iron=blue. Fröhde's reagent=violet. **Morphin.**

(*b*) Not colored by nitric acid or sesquichlorid of iron. **Cinchonin.**

bb. The solution becomes blue-violet on warming. **Papaverin.**

b. Sulphuric acid=grayish-brown, and on standing blood-red. **Narcein.**

c. Sulphuric acid=blue-violet. **Alkaloid of the celandine.**

X. Shake the ammoniacal fluid with amyl alcohol.

AMYL ALCOHOL RESIDUE FROM THE AMMONIACAL FLUID.

a. Sulphuric acid=colorless solution in the cold. **Morphin.**

b. Sulphuric acid=bright yellowish-red color, which becomes brownish. Iodin water=deep brown color. The alcoholic solution gelatinizes. **Solanin.**

XI. Evaporate the aqueous fluid with powdered glass, and extract the powdered residue with chloroform.

The residue from the first chloroform extract slows the respiration in a frog; that of the second and third gives a blue color, which changes to a permanent red when treated with sulphuric acid and potassium bichromate. Another part of this residue becomes red on heating with dilute sulphuric acid. **Curarin.**

The principles, as thus obtained by the above process, must then be subjected to the various tests mentioned under the head of each individual poison.

XI. Physostigmin (Calabar bean.)

774. **Properties.**—"The seed of Physostigma venenosum (U. S. P.) is about 25 to 30 millimeters long, 15 to 20 millimeters broad, 10 to 15 millimeters thick; oblong, and somewhat reniform; testa granular, chocolate-brown, with a broad, black groove extending over the entire length of the convex edge; embryo with a short curved radicle, and two large, white, concavo-convex cotyledons, inodorous; taste bean-like. On moistening the embryo with potassium hydrate T. S. it becomes pale yellow." (U. S. P. 1890, p. 298.)

775. **Preparations.**—Solid extract of Physostigma and tincture of Physostigma, 150 parts to 1,000. Physostigmin salicylate (eserin salicylate); physostigmin sulphate (eserin sulphate).

776. **Poisoning from use of.**—Mr. Blyth relates that in August, 1864, 46 children were poisoned at Liverpool by eating some Calabar beans which had been thrown on a rubbish heap which was part of a cargo from a ship from the west coast of Africa. One boy ate six beans and died. In April of the same year, two children, aged six and three years, ate the broken fragments of one bean; gastric irritation and muscular weakness followed, but both recovered.

Calabar beans have long been used by the superstitious tribes in West Africa as a test for innocence when a person is accused of theft; if innocent, it is supposed that the accused will vomit the drug, and live. Christison once took 12 grains of Calabar beans, and suf-

fered from torpor and giddiness with loss of muscular power. His heart and pulse became extremely feeble, and beat irregularly. Being alarmed at the serious symptoms, he took an emetic, and vomited; afterwards, he fell asleep, and the next day he was well.

777. Symptoms of poisoning.—These are similar, usually in warm-blooded animals, and begin with weakness in the voluntary movements, with peculiar muscular tremblings and twitchings,—especially of the lower part of the body,—and soon extend to the whole body; the animal falls to one side and cannot raise itself, though it may make strong efforts so to do. The saliva and tears are increased, and the intestines are the seat of violent catharsis: there is also vomiting. The respiratory movements are, at first, rapid and deep, as in nicotin poisoning; later, they become slow and difficult; the action of the heart is weak and slow, and the pupils are contracted to a very small point; intensity of these symptoms, as the poison is absorbed into the blood, increases until respiration ceases, the animal dying from asphyxia.

When the poison is administered to frogs, voluntary movements disappear very soon after its injection; respiration ceases, and the reflex response disappears.

778. Lethal dose.—Poisoning by Calabar beans is not very common, except from overdose, and then usually not fatal. Physostigmin undergoes decomposition except when closed in bottles, and soon loses its activity. The lethal dose, except in the case above named, where a boy, aged six years, ate six beans with fatal result, is not known.

779. Treatment.—The subcutaneous injection of scopolamin, with artificial respiration; perhaps, atropin.

780. Action.— This simulates very strikingly that of nicotin, *viz.*, it suspends or inhibits the reflex action in the central nervous system upon the movements of respiration and the heart's action. There is no local action. Apparently, the excitement of the spinal cord, medulla oblongata, and brain, afterwards the exhaustion of the functions of these centers, follow its administration, from the fact that repeated doses exaggerate the symptoms. It may be assumed that there is some interference with its elimination, so that continued doses may produce more serious results than one single dose of the same size; the so-called "cumulative" effects are thus produced. Physostigmin is excreted in the urine mainly, appearing in it a few minutes afterwards. It is also eliminated by the saliva and bile. The mucous secretions increase under its use and also the secretion of the pancreas. It seems to have a specially contracting action upon

the involuntary muscular fibers; as, for instance, it causes strong contractions of the bladder, uterus, and bronchial muscles. The intraocular muscles also undergo contraction. The pupils of the eyes also contract, whether physostigmin is employed externally or internally; this is controlled by atropin, though not entirely removed. The ciliary muscle is also acted upon in the same way as the pupils, so that the eye becomes accommodated for near distances. Its action resembles that of pilocarpin and muscarin. The intraocular pressure is considerably reduced with the application of physostigmin to the eye; Grünholm[69a] attributes this action to a contraction of the intraocular vessels, by which this secretion is diminished.

781. Chemical examination.—Physostigmin is slightly soluble in water, and easily soluble in alcohol, ether, chloroform, benzol, and amyl alcohol. The last three solvents extract it from aqueous alkaline solutions. It is easily soluble in dilute acids and alkalies. Its solutions are colorless; but, when exposed to the light, and still more quickly, if moderately warmed, they become a beautiful red color, owing to the formation of rub reserine. It is not precipitated from its solutions by platinic chlorid or picric acid. There are no characteristic color tests for physostigmin. The best test is the physiological, which consists in introducing a little of the solution into the eye, contraction of the pupil being the result. The same precautions should be taken as in performing the physiological test for atropin, which also causes dilatation of the pupil. Calabarin differs from physostigmin in being insoluble in ether.

782. Isolation from organic mixtures.—This is very difficult, on account of the ready decomposition of physostigmin in both acid and alkaline solutions by both heat and light, so that these influences must be avoided, as much as possible, in performing an analysis in cases of suspected physostigmin poisoning. The method of Dragendorff, as described under opium (see § 773) may be employed. Dragendorff has succeeded in detecting it in the contents of the stomach, intestines, saliva, and blood. By long contact with decomposing substances it is destroyed. Dragendorff was unable to detect it in blood which had been preserved for three months after death.[70]

XII. Pilocarpin.

783. Occurrence.—Pilocarpin is an alkaloid derived from the dry

[69a] Archiv. fur Opthalmol. XLIX. p. 620.

[70] Op. cit.

leaflets of Pilocarpus pennatifolius. Pilocarpus jaborandi, from which is made a tincture and fluid extract, has also been recognized by the British Pharmacopœia.

784. **Action.**—Pilocarpin has a powerful action in increasing the production of saliva and sweat; in our first chapter we have mentioned a peculiar case, in which the dead body of a patient, who had just before dying taken pilocarpin, had a post-mortem sweat upon the surface of the body. Pilocarpin seems to stimulate the motor nerves of the involuntary muscles, the contraction of the pupils, and an increased peristaltic action of the bowels.

785. **Symptoms of poisoning.**—First, a blushing of the surface, followed by copious perspiration, salivation, an increased secretion of mucus in the nose and bronchial tubes, and lachrymation; second, cardiac depression, pulse at first quickened, and distressed breathing; third, nausea, vomiting, and great thirst; fourth, pupils contracted, interference with the muscles of accommodation of the eye, fits, prostration, giddiness, occasional mental disturbance, which is generally accompanied with hallucinations.

786. **Lethal dose.**—No fatal poisoning has been recorded by pilocarpin; but, probably, 2 grains (0.13 gm.) would be a dangerous dose.

787. **Treatment.**—If the poison has been taken by the mouth, and not by subcutaneous injection, the stomach should be emptied mechanically, or by an emetic of mustard and water; atropin will counteract the effects of the poison when absorbed, and should be administered as an antidote, in doses of 1/15 to 1/30 of a grain (0.004 gm. to 0.002 gm.) of atropin sulphate, subcutaneously injected in successive doses until dilatation of the pupils is present and excessive sweating diminishes. Kobert states that pilocarpin should not be administered to weak and infirm old people; and never, unless atropin is at hand to check excessive action. The urine contains pilocarpin unchanged, after jaborandi leaves have been ingested.

XIII. Veratrum viride; veratrin.

788. **Occurrence.**—The white and the black hellebore, besides being violent irritants of the stomach and bowels, produce nervous symptoms, such as cramps, convulsions, and delirium. American or green hellebore has occasioned similar phenomena in a slight degree; but its poisonous action upon the nervous system is rather

shown by vertigo, somnolency or coma, dimness of sight, dilatation of the pupil, and impaired muscular action.

The poisonous action of these plants is due to the active principles contained in them; *viz.*, helleborin and helleborein called "glucosids;" and others called the "Veratrum alkaloids." The only one recognized as officinal, U. S. P., is veratrin, described therein as "a mixture of alkaloids obtained from the seed of Asagræa officinalis; a white or grayish-white, amorphous or semicrystalline powder, odorless, but causing intense irritation and sneezing when even a minute quantity reaches the nasal mucous membrane; having an acrid taste, and leaving a sensation of tingling and numbness on the tongue; permanent in the air."

789. Preparations.—Oleatum veratrinæ, unguentum veratrinæ, extractum veratri viridis fluidum, tinctura veratri viridis. Other alkaloids, nonofficinal, are the cevadin, the veratrin of Merck, crystallized veratrin in cevadilla (sabadilla) seeds; the veratrin of Wright and of Coürbe, "amorphous veratrin" in cevadilla seeds, also traces in Veratrum viride, and in veratrin (U. S. P.); jervin, in Veratrum viride (0.02 per cent) and in Veratrum album (0.13 per cent).

790. Alkaloids.—The roots of the Helleborus viridis, Helleborus fœtidus, and Helleborus niger owe their action to active principles which belong to the class of organic substances termed alkaloids. Helleborin, a glucosid from these plants derived by treating the roots with alcohol, is easily soluble in alcohol and chloroform, but with difficulty in water and ether; concentrated sulphuric acid added to it gradually produces a violet color. Helleborein, another alcoholic extract from the roots, is soluble in water, from which it can be precipitated by tannic acid and by the phospho-molybdate of sodium; it is with difficulty soluble in absolute alcohol and almost insoluble in ether; concentrated sulphuric acid produces with it, almost immediately, a beautiful, bright red color. Helleborein is the more important principle from a chemico-legal point of view, since it can be isolated from organic mixtures in precisely the same way as colchicin, and can be detected in the chloroform residue by the above tests (§ 773), and by the physiological effect which it produces when given to animals; since it is an intense heart poison, it diminishes strongly the frequency of the pulse beats. These glucosids, and the alkaloids as well, possess in a high degree the poisonous properties of the root of the Hellebores.

791. Symptoms of poisoning.—These are great nausea accompanied with pain in the stomach and bowels, violent vomiting, prostration,

cold extremities; small, slow, and feeble pulse; weak action of the heart; dizziness; blindness; dilatation of the pupils; sleepiness; coma, sometimes, with stertorous breathing. Death is due to asphyxia, and may occur, sometimes, in five or six hours, but generally after twenty-four hours or longer. Small doses reduce the force and frequency of the heart's action; but larger doses increase the rate of the pulse, which afterwards becomes feeble and irregular; the bodily temperature is reduced, and the general condition is one of collapse. Muscular trembling occurs in man, convulsions in animals, and, finally, paralysis, showing that it is a muscular poison. Physicians caution against its use as a remedy in pneumonia, except with robust patients and in asthenic forms of diseases. Its present use in medicine is mainly confined to ointments containing veratrin for the local treatment of neuralgia. The dose of veratrin (U. S. P.) as a medicine is 1/12 to 1/6 of a grain (.005 to .01 gm.) every three hours until the effects above described as resulting from small doses are observed, or until there is evidence of nausea.

Peterson and Haines quote four cases of symptoms of poisoning; in one, a child of eighteen months died in thirteen hours after 4 or 5 doses of 4 minims (0.26 c. c.) and then 1 dose of 16 minims (1 c. c.) of the tincture of Veratrum viride; in another, a woman of fifty years, after taking 70 minims (4.3 c. c.) of a fluid extract of Veratrum viride in two doses, died four weeks later. Serious symptoms of poisoning were shown in the other two cases of adults, after one had taken 1 dram (3.9 c. c.) of the tincture, and the other a teaspoonful of the fluid extract of Veratrum viride; but both recovered.

792. **Lethal dose.**—The fatal dose of the drug cannot be definitely stated, on account of the great difference in the susceptibility of persons to this agent. Serious symptoms of sedative action may be followed by a condition of collapse; should the medicine be suspended, the patient usually recovers with rapidity from a condition apparently dangerous. When administered under the careful eye of a physician, dangerous symptoms should not arise, unless by some mistake or accident, or an overdose be given.

793. **Treatment.**—Stimulants—especially morphin—are to be used in case of an overdose.

794. **Post-mortem appearances.**—The post-mortem appearances are not distinctive, though congestion of the lining of the stomach and bowels, and the passive congestion of the brain and its membranes may be observed; congestion, also, of the kidneys should be looked for.

795. Properties and tests.—A number of species of genus Veratrum have been found to contain alkaloids, the most important of which is veratrin, often called cevadin; and the other alkaloids are the veratrin of Wright and Coürbe, jervin, and still others of less importance. Of these, the first mentioned is the only one producing marked toxic symptoms, and which need concern us at present.

As veratrin is eliminated somewhat by the kidneys, special attention should be given to analyzing these organs and any urine that may be obtained in cases of suspected poison by this drug.

Veratrin is white, and crystallizes with difficulty from alcohol in the form of colorless prisms, which melt at about 115° C., and, at a higher temperature, volatilize. It is readily soluble in alcohol (about three parts) and chloroform (about two parts); it is also soluble in benzol, amyl alcohol, and ether (about 10 parts), but soluble with difficulty in naphtha and water; it is readily dissolved by dilute acids, salts of veratrin being formed, which are soluble in water, and from such solutions the veratrin is easily precipitated by alkalies and alkaline carbonates, into the amorphous form at first, the precipitate gradually becoming crystalline. The slightest amount of veratrin brought in contact with the nasal mucous membranes creates violent sneezing. Concentrated sulphuric acid dissolves veratrin, and forms a solution which is yellow at first, but changes gradually to an orange, then to a blood-red, and, after about one half hour, to a beautiful carmine-red color which is quite permanent; this change is hastened by heating or by exposure to the action of bromin vapor. If veratrin is first mixed with a little sugar, and then treated with concentrated sulphuric acid, the mixture is at first yellow, but quickly changes to a grass-green color, then to a beautiful blue, and, finally, to a dirty violet color; this change begins at the outside of the mixture and progresses inwards, and can be hastened by the addition of a little water (Weppen's test). "Concentrated hydrochloric acid dissolves the pure alkaloid without change of color; but, if the solution be heated to the boiling temperature, it quickly acquires a red color, which ultimately becomes very intense and resembles that of a solution of permanganate of potash. Under this reaction, if only a drop of the acid be employed, almost the least visible quantity of the alkaloid will manifest itself."[71] (Trapp's reaction.)

Upon evaporating a few drops of a solution of fused zinc chlorid

[71] Wormley, op. cit.

in dilute HCl to dryness with veratrin, a red color develops. (Schumpelitz.)

The physiological test for veratrin is an exceedingly important one in legal cases. The experiments of Weigelin[72] have shown that veratrin dissolved in very dilute acetic acid acts very energetically upon frogs, in a dose of 4/10 of a milligram; violent retching is produced very quickly, and the action of the heart becomes slower, the pulsations being diminished from 60 per minute to 32, after the lapse of ten minutes, and to 8, after the lapse of ninety minutes, when the heart's action becomes very irregular and sometimes ceases entirely. Tetanic convulsions are caused by larger doses, such as 2 milligrams; this dose produced immediate retching in medium-sized frogs; in fifteen minutes, tetanic spasms; and death in one hour.[72a]

796. **Isolation from organic mixtures.**—Veratrin can be isolated from organic mixtures by the process of Dragendorff, recommended for the opium alkaloids (see § 773). It is removed from the alkaline solution by shaking with naphtha or benzol, preferably the latter solvent; the residue left after the evaporation of the benzol can be used for performing the above tests.

797. **Difference in physiological action between veratrin and strychnin.**—The reader is referred to a previous page on strychnin, where the difference in the physiological action between veratrin and strychnin upon the muscular system of frogs is compared (p. 615).

[72] Dragendorff, op cit. p. 210.
[72a] See certain experiments by writer, in Bos. Med. & Surg. Journal, 1869.

CHAPTER VI.

PTOMAIN POISONING.

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801. In general.—Of recent years, numerous cases of poisoning with fatal results have been observed after the ingestion of various foods, chiefly meat or meat products, cheese, and milk.[1]

802. Occurrence and nature of ptomains.—Ptomains are substances of a basic character; they always contain nitrogen, and are often called putrefactive alkaloids. Since they may be formed from vegetable as well as animal substances, the term animal alkaloid is incorrect. As a rule, they are produced by the action of bacteria upon proteid material. All ptomains are not necessarily poisonous, although ordinarily considered so. There have been a few ptomains that have been isolated; namely, mytilotoxin, obtained by Salkowski and Brieger from mussels; tyrotoxicon, obtained by Vaughan; cholin, cadaverin and putrescin, isolated by Brieger; and sepsin, obtained by Faust. The nature of the ptomain depends upon the nature of the organism present, the kind of material, temperature, etc. As a rule, ptomains are formed in the early stages of the decomposition of meat or fish; therefore, material that has become rotten may be harmless, whereas, other material which does not even taste or smell of any decomposition may be poisonous.

In 1883–84, during a severe outbreak of sickness, caused by people eating poisonous cheese, in some three hundred cases Vaughan

[1] See Chap. I. § 3.

isolated the poisonous principle tyrotoxicon. The symptoms were abdominal pain, vomiting, diarrhea, weak and irregular pulse, and cyanosis. The same symptoms were observed in a number of persons who drank some milk. These cases were also investigated by Vaughan; the symptoms were severe vomiting, constriction in the throat, difficulty in swallowing, dilated pupils, and subnormal temperature. On examination of the premises where the milk had been kept, evidences of tyrotoxicon were obtained, both chemically and physiologically. In 1885, in Wilhelmshafen, Germany, there occurred an outbreak of poisoning, which was attributed to mussels; similar symptoms to those mentioned above were induced, except that there was no diarrhea or abdominal pain, and the legs became weak so that the patient could not stand. From these mussels Salkowski and Brieger isolated a body which they called "mytilotoxin." In most cases of ptomain poisoning, the symptoms are those that have been mentioned above. Post-mortem appearances are generally those of gastroenteritis.

803. Poisoning by toxalbumins, in general.—Cases of poisoning by toxalbumins may be classified under two headings, *viz.*, those produced by certain species of bacteria in food, where the food was originally wholesome; and those produced by toxins which were present in an animal before death. In the first case belong those cases known as "botulism." This is a poor name, as other food than sausages—especially fish—has been found to be the cause.

804. Poisoning by use of food which was originally wholesome, known as botulism.—These cases of botulism have been found to be due to the anaerobic bacterium, baccillus botulinus. Different species of proteus, however, also form toxins which have caused many cases of fatal poisoning.

805. Symptoms of botulism poisoning.—As a rule the onset is rapid, occurring in from twelve to twenty-four hours after the ingestion of the food. The symptoms are vomiting, diarrhea, abdominal pain, —even severe colic. They are often followed by constipation. Later, symptoms of poisoning of the central nervous system appear, with dilated pupils, loss of accommodation, and ptosis. There is more or less constriction in the throat, and dysphagia prevents the relief of great thirst. The skin is cold, and may be dry or moist. As a rule, the temperature is subnormal, the pulse small and rapid. Consciousness is retained generally to the last. The urine will often show evidences of acute renal irritation. Slight convalescence followed by relapse is common, and, in fact, a tendency to relapse is general.

The symptoms of poisoning by proteus are similar to those of the baccillus botulinus.

806. Post-mortem appearances from botulism poisoning.—The post-mortem appearances in cases of death from botulism show nothing very characteristic. There may be marked congestion of the organs, —especially of the lungs, liver, and kidneys. The mucous membrane of the stomach is often soft and congested.

807. Differential diagnosis between poisoning by toxalbumins and some of the alkaloids.—A differential diagnosis between poisoning by toxalbumins and poisoning by some of the alkaloids is not easily made. In poisoning by hellebore there are convulsions, stupor, collapse, and marked salivation; by conium, paralysis of the lower extremities followed by paralysis of the upper extremities; by belladonna, the onset is rapid, the face is flushed, and consciousness is lost early; by gelseminum, the course of the attack is much shorter, there is less visual disturbance, and the secretions are not affected.

808. Poisoning by toxalbumins in the body before the death of the animal.—Bolinger, in 1876, showed that a very large proportion of cases of food poisoning was due to the ingestion of the flesh of animals suffering from pyemia and septicemia. The reluctance of the poorer classes to suffer the loss of an animal through disease, when that animal could be used for food, has often led to the anticipation of the natural death of the animal by slaughter; and the meat is then used for food. Van Ermengem succeeded in isolating a bacillus, resembling Gaertner's bacillus enteritidis, from the bone marrow. In a number of cases, the bacilli of the coli group have been isolated, which produced toxins capable of withstanding high temperatures.

809. Symptoms.—Vomiting, diarrhea, abdominal pain, headache, prostration, fever, and chills are the usual symptoms; but these may be so severe as to produce the most profound disturbances, resembling symptoms of cholera and typhoid fever. Often, the cooking of the meat will prevent these symptoms; but they frequently occur among the poorer classes who are the most liable to exhibition of this form of poisoning when the food is eaten raw.

810. Treatment.—Rational treatment for cases of poisoning by ptomains or toxalbumins is, with the present knowledge of the subject, almost impossible, partly because the onset is so often delayed that removal of the poisonous material, which would ordinarily be the first object, is impossible. If the onset is rapid, and vomiting or purging has not occurred, these actions may be induced by gentle means; but if they have already occurred, it is useless to aggravate

the condition by treatment, so that methods of treatment must be governed by the individual symptoms as they arise.

811. Poisoning by use of vegetables.—The cause of poisoning by vegetables in this country has been chiefly attributable to ergot in rye and solanin in potatoes. The former has often been the cause of chronic poisoning in Europe where the rye forms a large part of the diet of the poorer classes. Many cases of acute poisoning by potatoes have occurred in Germany; the presence of the increased amount of solanin is due, probably, to the growth of two species of bacteria; *viz.,* bacterium solaniferum noncolorabile, and bacterium solaniferum colorabile.

CHAPTER VII.

EXAMINATION OF BLOOD, BLOOD STAINS, AND SEMINAL STAINS.

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I. Physiological and chemical properties of blood.

812. In general.—The blood consists of a liquid and a solid portion. The liquid is called the "blood plasma," and in it are suspended the red and white blood corpuscles and the blood plates, the solid portion. We also find granules; but, at present, we know very little concerning them, and they are as yet of no medico-legal importance. The blood has a slightly alkaline reaction, and the specific gravity varies from 1.058 to 1.062. In the human being the amount is roughly estimated at about 1/12 to 1/14 of the body weight. If we allow the blood to flow into a jar which has been cooled, and its contents are not disturbed, the liquid will separate into three layers, —an upper, light yellow layer of plasma, beneath which is a whitish layer of white corpuscles, and below this a wider layer consisting of red blood corpuscles. This plasma will readily coagulate if the temperature is raised above zero. If, on the other hand, we allow the blood to coagulate as soon as it leaves the body, it will form a solid mass, which, on standing, retracts from the walls of the vessel, leaving a clear amber-colored fluid which is called the "blood serum." The solid portion in this case is called the "blood clot," and consists of fibrin and the blood corpuscles. The difference, then, between the blood serum and the blood plasma is that the serum contains no fibrin, whereas, the blood plasma will coagulate, and, therefore, contains fibrinogen, the mother substance of fibrin. The blood plasma contains several proteid bodies, from which the fibrin separates upon coagulation of the blood. These proteids, so far as is known at present, are fibrinogen, serum globulin, and serum albumin. As to the exact nature and differences of these various proteids, we are not concerned here.

813. Blood serum.—As already mentioned, the blood serum differs from the plasma in not containing fibrinogen, but it does contain the fibrin ferment. It is more alkaline than plasma, of a specific gravity of about 1.028, faintly yellow in color, and contains, in addition to the substances already mentioned, lecithin, cholesterin, soaps, and sugar. The yellow coloring matter probably belongs to the luteins or fat coloring matters. The mineral bodies in serum and

in plasma are of the same kind, though different in amount. They are potassium, sodium, calcium, magnesium, chlorin, oxygen, and phosphoric acid.

814. Solid elements of the blood.—The solid elements of the blood are the red and white corpuscles, the blood plates, and the granules, although it is claimed that the last are not always present. Of these, the red cells are the most important, and, medico-legally, are practically the only ones that we have to do with. The red cells are the bodies that contain the coloring matter, the hemoglobin, and it is with these that we are chiefly concerned in examining blood stains. The number of red cells in the blood varies in different animals, and in different individuals. In normal, healthy male adults, there occur about 5,000,000 of these cells in each cubic millimeter. In females the number is slightly less, about 4,500,000. These red cells are heavier than the blood plasma or serum, and, therefore, they sink to the bottom of the vessel in which the blood is contained. The white cells, or "leucocytes," as they are called, occur in the human blood in about the proportion of 1 to 700 red cells. They are somewhat larger than the red cells, and their number may be greatly increased in disease; but, from a medico-legal point of view, they are practically of little importance. The blood plates and granules as yet are of no medico-legal interest.

815. Forms of the corpuscles.—The red corpuscles of the blood of vertebrate animals are generally one of two forms,—either circular or more or less oval. In all mammalia the corpuscles are nonnucleated, while in all oviparous animals they contain a nucleus; it is by this property that we are able to distinguish mammalian blood from that of birds, fishes, or reptiles. The size of the red blood cell differs more or less according to the animal, and also varies somewhat in the blood of the same animal, although close uniformity exists in the greater proportion of the red corpuscles of the same animal. With very few exceptions, the average diameter of the red corpuscles of mammals varies from 1/3000 to 1/6000 of an inch. The largest are those of the elephant, having a diameter of about 1/2750 of an inch, and the smallest those of the musk deer, measuring about 1/12325. In the blood of all birds, reptiles, and fishes, the red blood corpuscles contain a distinct nucleus, which is generally oval in shape, but may be nearly or quite circular. This nucleus is, as a rule, of darker color than the corpuscle itself, slightly irregular in contour, and generally occupies the central portion of the corpuscle. In the blood of birds, the nucleus is much more elongated than the corpuscle it-

self. The corpuscles of the oviparous class are considerably larger than those of mammals, the largest being those found in the blood of reptiles.

816. Coagulation of blood.—The physical phenomenon of coagulation has already been spoken of; but its importance is such as to demand a few more remarks. The time required for complete coagulation differs with the blood of different animals and under different circumstances; the time required may differ with the blood from the same animal.

817. Time of coagulation.—The blood of the fowl begins to coagulate in one and a half minutes, that of the rabbit and sheep in from one half to one and a half minutes, that of the dog in from one to three minutes, that of man in from three to four minutes, and that of the horse and ox in from five to thirteen minutes. The coagulation of human blood is generally complete in nine or ten minutes. The blood of oviparous animals coagulates more rapidly than that of cold-blooded animals, and the coagulum is much larger with the former. The coagulation of the blood may be hastened by slightly warming it, by its adhering to a rough surface, or by allowing it to stand in thin layers. Coagulation is retarded when the temperature is lowered, and it may even be prevented for hours if the blood is rapidly cooled to zero. Coagulation is also delayed when the blood comes in contact with greasy substances; it will generally be prevented by heating the blood quickly to a temperature of 56°, thereby destroying the action of the enzyme.

818. Slowness of coagulation as an aid in determining direction from which blood came.—In connection with this phenomenon of slow coagulation, the question of the possibility of determining the direction from which the blood came may be capable of solution. When the blood falls upon a horizontal surface in an oblique direction, the bulk of the blood is carried to a point away from its source; and, if the force is great enough, there may be one or more spatters or minute clots beyond. If the blood is thrown upward upon a vertical surface, the force may be enough to carry the blood to the top of the stain. If coagulation is slow, then the blood will tend to gravitate; but, if coagulation is rapid, some spots may be found with the greater portion of the blood at the top. In connection with this phenomenon of coagulation, there is also the drying of the blood to be considered. Blood dries much more slowly than water, and the character of the surface upon which the blood falls has considerable influence upon the time of drying. If the surface is a porous one,

drying takes place rapidly; but, if it is nonabsorbent, like glass or wood,—especially a planed surface,—the time of drying is much longer.

819. Color of blood stain.—The color of the blood stain varies a great deal with its age. At first, it is ordinarily a bright scarlet color; but, on exposure to daylight and to the air, the color becomes darker, changing to a dull brown. This change may be hastened by exposure to sunlight, or by heating the stain, as, for example, by ironing after washing the garment, or by the application of alcohol or other preservatives. From the above we see how necessary it is that the expert should carefully examine the stains as soon as possible after the receipt of any article to be examined for blood, in order that he may obtain as much information as possible in regard to the intervening time since the blood was spilled, the direction from which it came, and its source.

II. Tests for determining presence of blood, in general.

820. In general.—We come now to the more strictly chemical and biological tests for determining the presence of blood, and also for determining whether the stain, if it proves to be a blood stain, is caused by the blood from a certain kind of animal; in medico-legal practice this generally means the determining of whether or not the blood is human blood. The methods employed are the chemical, spectroscopical, microscopical, and biological tests. The first two are for the purpose of detecting the red coloring matter of the blood; this test will determine whether or not the stain is a blood stain.

After the stain has been determined to be a blood stain, the microscopical examination enables us to tell whether or not the blood is consistent with being human blood, and the biological test now enables us to express an opinion as to whether the blood is human blood or that of some other animal. The chemical tests have to do more especially with the hemoglobin of the red cells. The hemoglobin is the substance that carries oxygen to the tissues. With oxygen it forms two compounds, oxyhemoglobin and methemoglobin. In the former the combination is a very loose one, while in the latter the combination is quite stable. Solutions of oxyhemoglobin have a bright cherry-red color, such as is seen in arterial blood. In medico-legal investigations it is the hemoglobin, or one of its decomposition products, and also the serum albumin, that are tested for. The detection of the last, however, is only confirmatory, as it occurs in substances other than blood, such as the white of egg; the difference

between egg albumen and serum albumin is not as yet of medico-legal importance. Hemoglobin also unites with carbonic oxid, carbon dioxid, and nitric oxid, giving rise to different chemical compounds,—carbon monoxid hemoglobin, carbon dioxid hemoglobin, and nitric oxid hemoglobin. The carbon monoxid hemoglobin is of medico-legal importance in connection with poisoning from carbon monoxid or water gas; but it is of little importance in reference to the investigation of blood stains. Hemoglobin heated to 100°C. is decomposed into an albuminous body called "globin," which is coagulated, and into a pigment called "hematin." This decomposition can also be brought about by strong acids or alkalies, in which case the globin is not coagulated, but exists as an acid or alkali albuminate. Hematin is a brown pigment, and is of importance in chemical and spectroscopical examinations of suspected blood stains. Hematin may be deprived of its iron, in which case it is called hematoporphyrin. In this case the ordinary chemical tests for the detection of hematin will fail; also if a high degree of heat is applied to the stain, such as might be accomplished by passing a hot sadiron over the stain. In this latter case the stain may be fixed, so that ordinary solvents will not extract it from the fabric; but it can be dissolved out by concentrated acids. Alcoholic sulphuric acid will furnish a solution which can be employed for spectroscopic examination.

III. Chemical tests.

821. In general.—The principal chemical tests for blood are Teichmann's test, the sodium tungstate test, the guaiacum test, and the albumin test.

822. Teichmann's test.—Teichmann's test is the most important for blood pigment. If the stain to be examined has dried upon glass or some nonabsorbent fabric, it is sufficient to remove a portion on the point of a knife and transfer it to a glass slide. If, on the other hand, the stain has soaked into the fabric so that a fragment cannot be removed, a small portion can be scraped from the fiber with the point of a knife, and the dust can be collected upon a glass slide; or a bit of the fabric containing the stain can be soaked in a drop of water for some time, and this drop can be used for performing the test. The dust or small fragment of the blood clot upon the glass slide is then to be treated with a drop of water, and a minute crystal of common salt or potassium iodid may be added. This drop

is then evaporated to dryness by gently heating the residue, covered with a cover glass, and a drop of glacial acetic acid allowed to run in under the cover slip. The slide is then gently warmed until bubbles of gas begin to appear under the cover slip; the slide is then allowed to cool, and is examined for crystals of chlorid or iodid of hematin, commonly called "hemin crystals." These hemin crystals occur in characteristic, small, rhombic plates with a yellow or chocolate-brown color. They are often arranged in crosses or rosettes; and, if the amount of blood is large, the forms may vary in some parts of the preparation, some being pointed or even oval in outline.

Care should be taken not to apply too great heat either in evaporating the first drop or after the addition of acetic acid. In the first case, the hematin may be destroyed, and, in the latter, the crystals may be carried to the edges of the cover slip by the active boiling of the acetic acid. As stated above, if the stain has been subjected to a high degree of heat, hemin crystals will not be detected; this will also be the case if the blood stain has been subjected to prolonged action of various disinfectants or to direct sunlight.

823. Sodium tungstate test.—This test is often employed to detect blood, where the stain has been partially removed from the fabric by washing, or where the blood stain has been soaked in some fluid, and thus a greater portion of it has been removed. The fabric containing the remains of the stain is soaked in a small vessel containing distilled water, and a pinch of potassium iodid is added. In this way the remains of the stain are thoroughly soaked out into the solution. After soaking for a few hours, with frequent manipulation with a glass rod, the solution is poured off into a beaker, and the fabric again extracted. This is repeated two or three times with fresh portions of water and potassium iodid, until all the stain is removed. The united extracts are then filtered, the filtrate is strongly acidulated with acetic acid, and a few cubic centimeters of a saturated solution of sodium tungstate strongly acidulated with acetic acid are added. The precipitate, which may be quite bulky if there is much blood pigment present, is then boiled, and forms dense chocolate-colored flocculi. These are filtered off, washed with water, dried, and tested by the hemin test; or a portion may be dissolved in very dilute sodium hydrate, and examined spectroscopically for the bands of hematin in alkaline solution. If the precipitate, on the addition of sodium tungstate, is slight, the mixture, after boiling, should be allowed to settle completely, and, by careful washing in a

watch glass or on a glass slide, the residue can be used for the hemin test.

824. Guaiacum test.—A bit of the stain, or a few fibers of the stained cloth, are placed in a small evaporating dish, and a few drops of water are added to dissolve out the pigment as much as possible. A few drops of tincture of guaiacum are then added, and the mixture should remain colorless. If, now, a couple of drops of a solution of peroxid of hydrogen is added, a bright blue color will be produced if there are traces of blood pigment present. According to Wormley, this test will react in a solution of 1 part in 5,000.

Fallacies.—The following substances may produce this reaction: Glue, casein, many compounds of iron,—especially chlorid, acetate, and, possibly, hydrate, which is always present in iron rust. According to Huenefeld, iron sulphate, cupric sulphate, cupric nitrate, chlorid of gold and sodium, manganese dioxid, and potassium permanganate will give this test. Therefore, if this test is positive, it can be considered only as confirmatory, and the other tests must be applied before affirming that a stain contains blood pigment.

825. Albumin test.—This is only a confirmatory test, inasmuch as many substances not containing blood pigment give this test. It is performed, practically, in the first part of the hemin test, namely, when a solution containing albumin is heated to 65–80°, the solution becomes turbid, and small flocculi or coagula may be seen on the glass slide.

IV. Spectroscopic examination.

826. In general.—A spectroscopic examination of blood depends upon the fact that a solution of hemoglobin, or its compounds, or its decomposition products, gives characteristic absorption spectra, when examined with a spectroscope. If the amount of blood to be examined is large, an ordinary spectroscope will suffice; but in most cases the amount of material is so small that a microspectroscope, which is really a microscope fitted with a spectrum eyepiece, is necessarily employed.

827. Preparation of stain for spectroscopic examination. — The method of preparation may depend upon whether the blood stain or blood has undergone decomposition; if it has not, and the amount of material at hand is large, a portion of the stain can be soaked out in a little water or a dilute salt solution, and examined with a spectroscope. When the oxyhemoglobin bands can be recognized, this same solution should then be treated with a little ammonium sulphid

or Stokes' reagent, and a single band of hemoglobin should be obtained. If the stain is slightly decomposed, we may get a mixture of both the oxyhemoglobin and the hemoglobin spectra. If, on the other hand, the stain is decomposed, we shall fail to get any solution of the pigment by treatment with water, and we must obtain the result by the addition of either a little dilute sodium hydrate solution or glacial acetic acid. If we use an alkali, we should get a spectrum of hematin in alkaline solution, which can be changed by the addition of a little ammonium sulphid, into the spectrum of hemochromogen. If we use the glacial acetic acid, then we get the spectrum of hematin in acid solution. If the blood pigment has undergone decomposition into hematoporphyrin, we must treat a fragment of the material with concentrated sulphuric acid, and we can then get the spectrum of hematoporphyrin in acid solution, if the stain contains blood pigment. If the amount of material is very small, so that we have to use a microspectroscope, the following method suggested by Richardson is perhaps the most satisfactory:

"Procure a glass slide with a circular excavation in the middle, called by dealers a "concave center," and moisten it around the edges of the cavity with a small drop of dilute glycerin. Thoroughly clean a thin glass cover about ⅛ of an inch larger than the excavation; lay it on white paper, and upon this place the tiniest visible fragment of the freshly dried blood clot. This fragment will weigh from 1/50000 to 1/25000 of a grain. Then, with a cataract needle, deposit on the center of the cover near your blood spot a drop of glycerin about the size of a small period, and, with a dry needle, gently push the blood to the brink of your microspocic band, so that it may be just moistened by the fluid. Finally, invert your slide upon the thin cover glass in such a manner that the glycerined edges of the cavity in the former may adhere to the margins of the latter, and, turning the slide face upward, transfer it to the stage of the microscope. By this method it is obvious that we obtain an extremely minute quantity of strong solution of hemoglobin, whose point of greatest density, generally in the center of the clot, is readily found under a ¼-inch objective, and tested by the adjustment of the spectroscopic eyepiece. After a little practice, it will be found quite possible to modify the bands by the addition of sulphuret of sodium solution, as advised by Preyer.

"In cases of this kind, where the greatest possible economy, or even parsimony of the material, is needful, I would advise the following mode of procedure for proving, or corroborating your proof

of, the existence of blood, so that its presence in a stain may be affirmed with absolute certainty:

"From a suspected blood spot upon metal, wood, paper, muscle, or cloth, scrape with a fine, sharp knife two or three or more minute particles of the reddish substance, causing them to come near the middle of a large, thin cover glass. Apply in close proximity to them a very small drop of ¾ per cent salt solution, bring the particles of supposed blood clot to its edge, and proceed as I have already directed.

"After thus examining the spectrum of the substance, you may generally, by rotating the stage, cause the colored fluid partly to drain away from the solid portion, wherein, under favorable circumstances, should the specimen be blood, the granular white blood globules become plainly visible, as do also cell walls of the red disks. Among the latter, if your mental and physical visions be keen enough, you can, by the aid of a 1/25 immersion lens and an eyepiece micrometer, measure a series of corpuscles accurately enough to discriminate human blood from that of an ox, pig, horse, or sheep."

828. Oxyhemoglobin.—The characteristic spectrum of oxyhemoglobin is best seen in very dilute solutions. In concentrated solutions the characteristic band is seen as one broad band extending from D to b. If this solution is diluted, the band is resolved into two bands, one lying close to the line D, which is darker, narrower, but more distinct than the other, which lies near the line E. On further dilution, the second band, that is, the one towards E, disappears, leaving only the one near D.[1]

829. Hemoglobin.—If the oxygen is removed from the oxyhemoglobin, either by exhaustion or by the action of some reducing agent, as Stokes' reagent, or a dilute solution of ammonium sulphid, it will have a reddish or bluish-red color, which is distinctly darker than the color of oxyhemoglobin, being practically the color of venous blood. The absorption spectrum of hemoglobin extends from D towards E, occupying the most of the space occupied by that of a strong solution of oxyhemoglobin; but the band is not so dark. It is better described as occupying the space between the two bands of a dilute solution of oxyhemoglobin.

[1] There are but few substances whose solutions give a spectrum at all resembling that of oxyhemoglobin, such as, solutions of alkanet root in alum, and those of cochineal. Therefore, the spectroscopic examination must not be confined to the recognition of the spectrum of oxyhemoglobin only. Different reagents must be employed, so as to obtain several of the blood spectra, inasmuch as the vegetable solutions just mentioned are not changed by the action of reducing agents.

830. Methemoglobin.—This is a compound of hemoglobin with oxygen and is much more stable than oxyhemoglobin. It is sometimes found in the living body in cases of potassium chlorate poisoning, and also in old blood stains that have been exposed to the sunlight for a long time. Its absorption spectrum shows a band between C and D, lying a little nearer C than D, and the two bands of oxyhemoglobin between D and E.

831. Carbon monoxid hemoglobin.—This is a very stable compound formed by the union of CO with hemoglobin or oxyhemoglobin. It is of medico-legal importance in cases of poisoning by illuminating gas rather than as a test for blood in stains. A solution of carbon monoxid hemoglobin has a very bright cherry-red color, which is much more persistent than the color of oxyhemoglobin, and it is much more resistant to reducing agents than oxyhemoglobin. Its absorption spectrum is practically identical with that of oxyhemoglobin, except that the two bands between D and E are slightly nearer the blue, and are not easily replaced by treating the solution with ammonium sulphid, as is the case with solutions of oxyhemoglobin.

832. Hematin.—The importance of the spectroscopic examination for hematin has been outlined above, namely, in those cases where the hemoglobin has been decomposed by various agencies. In order to prepare it for spectroscopic examination, it is extracted either with a dilute alkali or glacial acetic acid, or an alcoholic solution of sulphuric acid. In the one case we obtain an alkaline solution of hematin, and in the other an acid solution. The alkaline solution has an absorption spectrum extending to the band between C and D, lying near D, often overlapping it, while the violet end of the spectrum is nearly absorbed. The acid solution has a band between C and D, near the line C. If we use alcoholic sulphuric acid, we may get four bands, one between C and D, near C; two bands between D and E, one near D, the other, darker, broader, near E; and a fourth between b and F.

833. Reduced hematin or hemochromogen.—An alkaline solution of hematin treated with a reducing agent, such as ammonium sulphid, gives a reduced hematin or hemochromogen. Its spectrum consists of a well-defined line between D and E and a fainter one between b and E.

834. Hematoporphyrin.—Blood pigment dissolved in concentrated sulphuric acid gives rise to hematoporphyrin. Its absorption spectrum has two bands, a faint one to the left of D, and the other between D and E. In alkaline solution, hematoporphyrin has four

bands, a faint one between C and D, two between D and E similar to the oxyhemoglobin bands, and a fourth one between b and F.

V. Microscopic examination of blood.

835. Method of preparation for examination.—In the preparation of dried blood for microscopic examination and measurement of the red corpuscles, it is necessary to bring the corpuscles into such a condition that they may be recognized by their normal shape, and to have them as free as possible from surrounding material, so that they can be properly focused. To do this it is necessary to use some solvent which will soften the blood and allow the corpuscles to separate. This menstruum must be of such a nature that it will not destroy the corpuscles or alter their shape, when it is proposed to measure the corpuscles. When it is a question simply of determining whether or not there are any blood corpuscles present, most any menstruum can be used. The principal menstrua that can be used are the following: Roussin's—glycerin, 3 parts; sulphuric acid, 1 part; and enough water to make the specific gravity of the liquid 1.028. Pacini's—water, 300 parts; glycerin, 100 parts; common salt, 2 parts; and corrosive sublimate, 1 part. Richardson's—¾ of 1 per cent common salt, after previously washing out the coloring matter with a dilute salt solution. Virchow's—30–33 per cent solution of potassium hydrate. Wormley recommends simply a small quantity of distilled water, approximately the amount originally present in the dried specimen. If long soaking is required, as in old stains, a dilute solution of glycerin (sp. gr. 1.030) may be used; and, if this is not sufficient, a little potassium hydrate may be added to the glycerin or water. Wood recommends the use of a solution of potassium acetate (sp. gr. 1.030) with the addition of a little formalin to prevent the formation of any fungus growth.

Having determined what menstruum is to be used, a portion of the dried stain is selected for microscopic examination. The portion selected must be where the blood corpuscles have dried quickly, otherwise they will have become more or less spherical, in which case they are unfit for measurement. If the blood has been dried in a thin layer upon some nonporous surface, such as glass or wood, a thin film may be scraped from the surface with a sharp knife on to a glass slide, and the cells may be fixed by treating them with a mixture of equal parts of absolute alcohol and ether. A drop of the solvent is then added, and a thin cover glass placed upon it and sealed so as to prevent evaporation. In the course of a longer or a shorter time, the mass will become softened, and the red corpuscles may be seen

under natural conditions. In case the blood stain or clot is upon an absorption material, such as cloth, much greater difficulty will be encountered in obtaining satisfactory results. On account of the capillary attraction in the meshes of the fabric, the corpuscles are more or less distorted, so that only rarely can one be found suitable for measurement. If, however, one can find a minute clot or thin crust, he may expect to find a sufficient number of corpuscles, the average measurement of which may be satisfactory. The finding of such crusts or minute clots occurs more often where the blood has formed upon material or substances which are firm, although slightly absorbed, such as felt. As a rule, the longer the clot has been dried before examination, the longer will it be necessary to soak the material before any satisfactory results can be obtained. If the fabric or garment has been carefully washed, of course all evidence of blood stain may have disappeared; but this, as a rule, is only accomplished by long soaking and careful rubbing in cold water, so that it often happens that the attempted rapid removal of blood from a garment leaves behind it more or less stain, which can be detected upon careful examination. Careful examination must be made to determine the presence of other substances, such as hair, muscle, bone, etc., as they may possibly throw some light upon the source of the blood.

As has already been noted, the blood cells of different animals differ in size and shape, and as to whether they are nucleated or non-nucleated. The question whether the blood is that of mammalia or that of bird or fish is easily settled, as the nuclei of bird blood are even more resistant than the cell, and can be easily recognized. The distinction between different kinds of mammalian blood, is, however, much more difficult, as the difference is practically a difference in the size of the red blood cells. This difference lies between very narrow limits, and a positive statement as to the kind of blood is often impossible. In the microscopic examination of blood stains, one is not required to state what kind of blood it is. He is only required to make a complete investigation, to state what his results are, and with what they are consistent.

In very accurate measurements for red blood cells, it is necessary to use the highest powers of the microscope, with a magnification of at least 1,000 to 1,500 diameters. The Jackson micrometer eyepiece is perhaps as good an instrument as any. Each micrometer scale must be accurately standardized before being used.

836. Average diameter of blood cells of man and other animals.—In medico-legal practice it becomes a question, as a rule, whether the

blood corpuscles are those of man or those of some domestic animal. The average diameter of the blood cells of the most common domestic animals, as well as those of man, falls within the limits of 1/3000 to 1/6000 of an inch. As will be seen from the following table, the various authorities differ only slightly with reference to the average diameter of the human blood corpuscles. It lies between 1/3300 and 1/3200 of an inch. The horse, cow, cat, dog, pig, sheep, and goat are the most common domestic animals whose blood has to be differentiated from that of man. Of these, the dog approaches most nearly the human, the average diameter of a blood cell being 1/3550 of an inch. The following table gives the average measurements as reported by various investigators. The figures given are in common fractions of an inch; this form is more commonly employed than the metric system, which, as a rule, is unintelligible to the average man:—

Average Size of the Red Blood Cells.

	Gulliver [1a]	Wormley [1a]	Formad	Richardson	C. Schmidt, 1848	French Medico-legal Society, 1873	Masson	Dragendorff	Woodard
Man	1-3200	1-3250	1-3200	1-3224	1-3330	1-3257	1-3256	1-3300	1-3092
Monkey	1-3412	1-3382	------	1-3395	------	------	------	------	------
Opossum	1-3557	1-3145	------	------	------	------	------	------	------
Guinea pig	1-3538	1-3223	1-3400	------	------	------	1-3300	------	1-3213
Kangaroo	1-3440	1-3410	------	------	------	------	------	------	------
Musk rat	1-3550	1-3282	------	------	------	------	------	------	------
Dog	1-3532	1-3761	1-3580	1-3542	1-3650	1-3479	1-3577	1-3628	1-3246
Rabbit	1-3607	1-3653	1-3662	------	1-3968	1-3681	1-3628	1-3968	------
Rat	1-3754	1-3652	------	------	1-3968	------	------	------	------
Mouse	1-3814	1-3743	------	------	1-4166	------	------	------	------
Pig	1-4230	1-4268	1-4250	1-4230	1-4098	1-4233	1-4098	1-4098	------
Ox	1-4267	1-4219	1-4200	1-4267	1-4385	1-4535	1-4233	1-4385	------
Horse	1-4600	1-4243	1-4310	------	1-4456	1-4535	------	------	------
Cat	1-4404	1-4372	------	------	1-4535	1-3907	1-4456	1-4535	------
Elk	1-3938	1-4384	------	------	------	------	------	------	------
Buffalo	1-4586	1-4351	------	------	------	------	------	------	------
Wolf (prairie)	1-3600	1-3422	1-3450	------	------	------	------	------	------
Bear (black)	1-3693	1-3656	------	------	------	------	------	------	------
Hyena	1-3735	1-3644	------	------	------	------	------	------	------
Squirrel (red)	1-4000	1-4140	------	------	------	------	------	------	------
Raccoon	1-3950	1-4084	------	------	------	------	------	------	------
Elephant	1-2745	1-2738	------	------	------	------	------	------	------
Leopard	1-4319	1-4390	------	------	------	------	------	------	------
Hippopotamus	1-3429	1-3560	------	------	------	------	------	------	------
Rhinoceros	1-3765	1-3649	------	------	------	------	------	------	------
Whale	1-3099	------	------	1-3090	------	------	------	------	------
Tapir	1-4000	1-4175	------	------	------	------	------	------	------
Lion	1-4322	1-4143	------	------	------	------	------	------	------
Ocelot	1-4220	1-3885	------	------	------	------	------	------	------
Mule	------	1-3760	------	------	------	------	------	------	------
Ass	1-4000	1-3620	------	------	------	------	------	------	------
Ground squirrel	------	1-4200	------	------	------	------	------	------	------
Bat	1-4175	1-3966	------	------	------	------	------	------	------
Sheep	1-5300	1-4912	1-5000	1-5300	1-5649	1-5080	------	1-5649	------
Ibex	------	1-6445	------	------	------	------	------	------	------
Goat	1-6366	1-6189	1-6100	------	------	------	------	------	------
Sloth	1-2865	------	------	------	------	------	------	------	------
Platypus (duck-billed)	1-3000	------	------	------	------	------	------	------	------
Capybara	1-3190	1-3164	------	------	------	------	------	------	------

[1a] Wormley, op. cit. p. 733.

Average Size of the Red Blood Cells.—Continued.

	Gulliver [1a]	Wormley [1a]	Formad	Richardson	C. Schmidt, 1846	French Medico-legal Society, 1843	Masson	Dragendorff	Woodard
Seal	1-3281	------	------	------	------	------	------	------	------
Woodchuck	1-3484	------	------	------	------	------	------	------	------
Musk deer	1-12325	------	------	------	------	------	------	------	------
Beaver	1-3325	------	------	------	------	------	------	------	------
Porcupine	1-3369	------	------	------	------	------	------	------	------
Llama (long diam.)	1-3361	1-3201	------	------	------	------	------	------	------
(short ")	1-6229	1-6408	------	------	------	------	------	------	------
Camel (long diam.)	1-3123	1-3331	------	------	------	------	------	------	------
(short ")	1-5876	1-5280	------	------	------	------	------	------	------
BIRDS									
Chicken (long diam.)	1-2102	1-2080	------	------	------	------	------	------	------
(short ")	1-3436	1-3483	------	------	------	------	------	------	------
Turkey (long ")	1-2045	1-1894	------	------	------	------	------	------	------
(short ")	1-3598	1-3444	------	------	------	------	------	------	------
Duck (long ")	1-1937	1-1955	------	------	------	------	------	------	------
(short ")	1-3424	1-3504	------	------	------	------	------	------	------
Pigeon (long ")	1-1973	1-1892	------	------	------	------	------	------	------
(short ")	1-3643	1-3804	------	------	------	------	------	------	------
Goose (long ")	1-1836	------	------	------	------	------	------	------	------
(short ")	1-3839	------	------	------	------	------	------	------	------
Quail (long ")	1-2347	------	------	------	------	------	------	------	------
(short ")	1-3470	------	------	------	------	------	------	------	------
Dove (long ")	1-2005	------	------	------	------	------	------	------	------
(short ")	1-3369	------	------	------	------	------	------	------	------
Sparrow (long ")	1-2140	------	------	------	------	------	------	------	------
(short ")	1-3500	------	------	------	------	------	------	------	------
Owl (long ")	1-1763	------	------	------	------	------	------	------	------
(short ")	1-4076	------	------	------	------	------	------	------	------
REPTILES									
Tortoise (long diam.)	1-1252	1-1250	------	------	------	------	------	------	------
(land) (short ")	1-2216	1-2200	------	------	------	------	------	------	------
Turtle (long ")	1-1231	------	------	------	------	------	------	------	------
(green) (short ")	1-1882	------	------	------	------	------	------	------	------
Boa con- (long ")	1-1440	1-1245	------	------	------	------	------	------	------
strictor (short ")	1-2400	1-2538	------	------	------	------	------	------	------
Viper (long ")	1-1274	------	------	------	------	------	------	------	------
(short ")	1-1800	------	------	------	------	------	------	------	------
Lizard (long ")	1-1555	------	------	------	------	------	------	------	------
(short ")	1-2743	------	------	------	------	------	------	------	------
BATRACHIANS									
Frog (long diam.)	1-1108	1-1089	------	------	------	------	------	------	------
(short ")	1-1821	1-1801	------	------	------	------	------	------	------
Toad (long ")	1-1043	------	------	------	------	------	------	------	------
(short ")	1-2000	------	------	------	------	------	------	------	------
Triton (long ")	1-848	------	------	------	------	------	------	------	------
(short ")	1-1280	------	------	------	------	------	------	------	------
Proteus (long ")	1-400	------	------	------	------	------	------	------	------
(short ")	1-727	------	------	------	------	------	------	------	------
Amphiuma (long diam.)	1-363	1-358	------	------	------	------	------	------	------
tridactylum (short ")	1-615	1-622	------	------	------	------	------	------	------
FISHES									
Trout (long diam.)	1-1524	------	------	------	------	------	------	------	------
(short ")	1-2460	------	------	------	------	------	------	------	------
Perch (long ")	1-2099	------	------	------	------	------	------	------	------
(short ")	1-2824	------	------	------	------	------	------	------	------
Pike (long ")	1-2000	------	------	------	------	------	------	------	------
(short ")	1-3555	------	------	------	------	------	------	------	------
Eel (long ")	1-1745	------	------	------	------	------	------	------	------
(short ")	1-2842	------	------	------	------	------	------	------	------
Lamphrey (circular)	1-2134	------	------	------	------	------	------	------	------
Lamphrey (diam. of nucleus)	1-6400	------	------	------	------	------	------	------	------

[1a] Wormley, op. cit. p. 733.

VI. Biological test for blood.

837. In general.—During the past eight or ten years, a great deal of attention has been devoted to the study of immunity in various diseases. This has naturally led to a study of the blood and the blood serum, and there have been found evidences of various specific toxins, and also various antitoxins, and certain other bodies with precipitating actions, called "precipitins," in the blood.

If one injects into a rabbit, either intraperitoneally or subcutaneously or intravenously, the blood serum of another species of animal, the serum of the rabbit so treated will produce a precipitate, when mixed with the serum of the animal whose serum was injected into the rabbit. Several kinds of animals will also react to this test, as well as a rabbit; but it has been found that rabbits are the best suited for this kind of work. For purposes of medico-legal examination of blood, namely, determining whether the blood may or may not be human blood, the rabbit is given injections of about 10 cubic centimeters of human serum at intervals of two or three days, and then allowed to rest a few days. Rabbits vary a great deal as to their susceptibility to the injection. Some apparently thrive on the treatment, and others are easily made sick. Further, rabbits vary a great deal in the amount of the precipitin which is produced in their sera. Some produce a very strong precipitin, others only a weak one, and, occasionally, they do not seem to react even after a dozen injections. In place of blood serum, any pleuritic effusion or exudate or hydrocele fluid may be used; but the serum itself is the best. When the rabbit has been treated as above, it can then be bled, either by killing the animal and taking the blood from the heart or from the carotid arteries, or, if it is desired to save the animal as a source of material, it can be bled from the central vein at the base of the ear. This last method is perhaps the easiest, as it does not necessitate the sacrifice of the animal, and sufficient blood, even with one bleeding, can be obtained to enable one to perform many control tests. This, of course, should be done under strict surgical asepsis. The blood, after being withdrawn, is allowed to coagulate; and, if necessary, it can be centrifugalized, and a clear, almost colorless liquid can be obtained. This serum will, if human blood was used for injection into the rabbit, react only with human blood, under proper precautions.

838. Method of examination.—If fresh blood is to be examined, it can be diluted with about 1 to 100 parts of normal salt solution, and, if necessary, allowed to settle so that the solution to be tested

is absolutely clear. If the stain is to be tested, and it has not soaked into the material, a few fragments of the clot can be scraped on to a slide or into a small test tube, and treated with a few drops of water, and allowed to dissolve. If the stain is upon a fabric, where it has penetrated the meshes of the cloth, then a few threads can be soaked out in a few drops of distilled water. In either case the material is diluted with salt solution. The degree of dilution is not of so much importance as is the degree of dilution of the serum obtained from the rabbit. However, a dilution of 1 part in 100 of the fluid to be tested seems to be the most favorable for the reaction.

The dilution of the rabbit serum is of more importance, as it is upon its dilution that the most careful and accurate results are obtained. Positive reactions have often been obtained with humanized rabbit serum and the sera of lower animals, when the rabbit serum was used in too great quantity. For example, in a proportion of 1 of rabbit serum to 10 of blood solution, or, in a proportion of 1 to 20, precipitates have been formed which might have been interpreted as positive. On the other hand, it has happened that with humanized rabbit serum, used in a proportion of 1 to 10, or 1 to 20, of solution of human blood, no reaction has been obtained; whereas, if the rabbit serum was used in the proportion of 1 to 50, or 1 to 100, a positive reaction resulted. A dilution of 1 of the test serum to 100 of the blood solution gives the most positive results. If the amount of material to be tested is small, this dilution may fail, and it may be better to use a dilution of 1 to 50. Another point to be borne in mind is the length of time that is to be allowed for the precipitate to occur. In using a dilution of 1 to 100, it often happens that the precipitate does not occur for an hour and a half or two hours; and, where the material available for the test has been small and old, even this length of time is not sufficient for the formation of the characteristic flocculent precipitate, and it may be even necessary to allow as much as six or twelve hours at a temperature of 37° C. So that, in dealing with minute stains and old ones, the results of the reaction must be interpreted according to the experience of the investigator, and, at best, can only be expressed as an opinion.

For a more detailed account of this test and its application to medico-legal practice, one is referred to the résumé of Professor Ewing,[2] and also to the recent work of Professor Nuttall.[3]

[2] Ewing & Strauss, Medical News, Nov. 7 & 14, '03.

[3] Blood Immunity and Relationships, G. H. F. Nuttall, University Press, Cambridge, 1904.

839. Conclusion.—In conclusion, one may say that it is often possible to distinguish between stains caused by human blood, and those caused by the blood of domestic animals; that, under favorable circumstances, it can always be determined whether the blood is mammalian or oviparous; that, if the blood is mammalian blood, and the cells have an average diameter of between 1/3300 and 1/3100 of an inch, that the blood is not that of a cat, goat, pig, sheep, horse, or ox, that it may be the blood of a dog, guinea pig, rabbit, or man; but most experts agree that one is not justified in saying that the blood is human blood. The most that an expert can say is that, if the blood cells have a diameter of 1/3200 of an inch, slightly more or less, that then these measurements are consistent with the blood being of human origin; that if the biological test, under favorable conditions, and properly performed by a recognized competent expert, gives the characteristic reactions, that the blood may then be said to be human blood.

The conclusion reached by Graham-Smith and Sanger [3a] lays down the conditions under which reliance can be placed in this biological test: "These experiments have led us to the conclusion that, with sufficient materials, and due precaution to exclude the various sources of error, there are but few conditions met with in forensic practice under which human could not be readily differentiated from other bloods. By this, however, we do not mean to imply that a considerable acquaintance with the action of precipitating antisera on blood solutions is not necessary in the successful application of this test."

That this test will be used in the future is also shown, to quote from Nuttall's book:[4]

"That this conclusion is fully justified is proved by the official recognition of the precipitin method in forensic practice in foreign governments. Prof. Uhlenhuth informs me that the method has been recommended by the Ministers of Justice in Germany and Austria, and that it has been officially recognized by the governments of Egypt and Roumania."

In the United States it has been used by Prof. Wood in the Greenleaf[5] and Blondin[6] cases.

VII. Stains containing blood in various discharges.

840. In general.—That the stain which has been proved to contain

[3a] Jour. Hygiène, 1903, Vol. III. No. 2, p. 260.
[4] Loc. cit. p. 403.
[5] Wood, Trans. Mass. Med.-Leg. Soc. 1902, Vol. III. No. 4.
[6] Blondin case, Suffolk Co. Mass. Dec. 1902.

blood may have been of menstrual or vaginal origin, or from some other source, can only be determined by finding, associated with the blood, other formed elements, such as epithelial cells, mucus, spermatozoa, pus, etc.

841. Menstrual blood.—As a rule, blood of menstrual origin will have vaginal cells in large numbers mixed with it. This is especially true at the beginning and at the end of the flow. If the hemorrhage is abundant it is possible that a stain—especially a small one—will not show any cells mixed with it; but we are not warranted in saying that the blood was not of menstrual origin, if we fail to find vaginal cells mixed with it. The location of the stain, whether upon underclothing or bedding, may be of importance in helping to recognize a stain as being of menstrual or lochial origin. Vaginal cells are large, polygonal, squamous epithelial cells, similar to those of the mouth, but with a nucleus of slightly larger diameter, often arranged in small clumps. If, at the time of the menstrual flow, there was also a discharge of gonorrheal or leucorrheal origin, then the blood and cells will be found mixed with pus. Occasionally, also, a ciliated cell from the lining membrane of the uterus may be found mixed with blood of menstrual origin.

842. Nasal stains.—In case of hemorrhage from the nose, identification of the source of the blood must be made by the recognition of the cylindrical or ciliated epithelium from the mucous membrane, and of dried mucus mixed with the blood. However, it is often the case that the hemorrhage is so severe that there may be stains which consist of blood only, and we cannot find either mucus or epithelium to help us to ascertain its source. If we find the cells and mucus, we can give an opinion that the blood is consistent with having come from the nose; but the reverse is not true—we are not justified in saying that the absence of mucus or cells rules out the possibility of the stain being of nasal origin. Here the position and other characteristics of the stain may be of help in forming an opinion, and enabling us to decide whether or not it is possible that the blood was of nasal origin. If the blood was caused by forced expiration through the nose the stain is apt to be pale and bulky, as the relative amount of mucus is considerable, and, on drying, the appearance is very different from that of an ordinary blood stain.

VIII. Seminal stains.

843. In general.—The examination of seminal stains does not, as a rule, include the great variety of substances that examination for

blood stains includes. The usual articles submitted for examination are bedclothes and underclothing; occasionally, other articles of wearing apparel; less often, bits of wood or earth or leaves; and also, occasionally, scrapings of dried masses taken from the skin or genitalia of the victim. Seminal stains may exist as dried masses of seminal fluid, or of this mixed with other fluids, such as blood, or discharges from the vagina.

844. **Properties.**—The seminal fluid varies according to circumstances, and at different times in the same individual. Its specific gravity varies from 1.027 to 1.037; it has a slightly alkaline reaction, is more or less viscid, with an opalescent appearance, sometimes grayish, occasionally yellowish or even reddish, though not mixed with blood. It also has a peculiar odor, which used to be considered more or less diagnostic, but this property has now lost its importance. When a seminal stain dries upon a nonabsorbent surface, such as wood or iron, or upon cloth or linen that has been starched, it forms a grayish scale, from which a bit can be removed by the point of a knife or needle. The grayish color may also have a faint yellowish or reddish tint. If the stain is upon a very porous or absorbent material, it may be very difficult to see. It will have a more or less irregular outline and a peculiar feel, which is more or less stiff, similar to that caused by blood or any albuminous fluid. It may often best be seen by holding it against transmitted light, when the stain will appear more or less translucent. The location of the stain varies often. It is more apt, in cases of alleged rape, to be found on the posterior portion of the victim's garments, or upon the anterior part of the assailant's clothing. The seminal fluid probably does not contain serum albumin. It does contain, however, cholesterin, lecithin, nuclein, cerebrin, guanin, and hypoxanthin. Schreiner[7] has isolated a substance called "spermin," C_2H_5N, which gives the general alkaloidal reactions. This body is also found in the sperm of several animals, also in the blood and sputum.

Dr. Florence[8] claims to have isolated from the seminal fluid of man an alkaloidal substance which he claims to be obtained only from human seminal fluid. This body, which he calls "virispermin," gives characteristic crystals with a concentrated solution of iodin in potassium iodid. This test is extremely delicate. Other investigators have claimed that any tissue containing cholin will yield these

[7] Schreiner, Annal. d. Chem. u. Pharm. Bd. 194.

[8] Florence, Du Sperme et des Taches de Sperme en Médecine Légale, 1897.

same crystals. The reagent known as Florence's reagent is potassium iodid, 1.65 grams; iodin, 2.54 grams; water, 30.00 grams.

To perform the test, it is necessary to proceed as follows: A small bit of the cloth or material containing the stain to be tested is cut away with scissors, and deposited upon a glass slide. A drop of water is then added, and the stain is allowed to soak a few minutes. A drop of the reagent is then placed beside the first drop, and the two are allowed to mix, and are immediately covered with a cover glass. If the stain contains seminal fluid, the appearance of crystals is almost immediate.

845. **Detection.**—The Florence test, at the best, is a good preliminary test. For the absolute determination that a suspected stain is a seminal stain, it is necessary to obtain the spermatozoa. These are bodies with an oval pear-shaped head, about 1/3000 of an inch in length, and a tail 9–10 times as long. The head is less dense in its anterior third than in the posterior two thirds. Spermatozoa are fairly stable, and resist decomposition; but, after drying, they are very fragile, and the head is easily broken from the tail, so that to obtain perfect spermatozoa is often difficult,—especially in an old stain. Staining, as suggested by Florence, helps in the recognition of the spermatozoa. Florence uses crocein, but methyl green can be used as well.

For the preparation of the stain for the microscopical examination, a small bit of the cloth containing the stain is soaked in distilled water on a slide, and then carefully teased apart with fine needles, covered with a cover glass, stained or not as preferred, and then examined with the microscope. In examining a stain that has soaked into a porous surface, such as unstarched cloth, the Florence reaction may often be obtained from any portion of the stain; but, as a rule, the spermatozoa will only be found in the center.

846. **Biological test.**—Dr. C. G. Farnum[9] has proposed the biological test along the lines as described under the section on blood. He injects into the peritoneal cavity 5 to 10 cubic centimeters of semen or testicular emulsion, at intervals of three or four days, until the rabbit has received five to eight injections. He has found the test specific for human semen when human semen was used for injection; and he does not get a reaction with the semen of the dog, bull, or goat, nor does human blood serum give a positive reaction.

[9] Am. Jour. Med. Asso. Dec. 28, 1901.

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APPENDIX.

Appendix, in which are recited the two laws concerning the medical examination required by the state of Massachusetts and the state of Connecticut in case of death from supposed violence. Also reports, from medical literature, of certain noted cases of poisoning, and abstracts of same.

847.—THE REVISED LAWS OF THE COMMONWEALTH OF MASSACHUSETTS, CHAPTER 24.

Enacted November 21, 1901, To Take Effect January 1, 1902.

Sec. 1. The governor, with the advice and consent of the council, shall appoint, for a term of seven years, able and discreet men, learned in the science of medicine, to be medical examiners and associate medical examiners in each county.

Sec. 2. The number of medical examiners and associate medical examiners shall be as follows: (This list by counties is omitted.)

Sec. 3. The associate medical examiner of the county of Suffolk shall, at the request of either of the medical examiners for the county, perform the duties and have the powers of a medical examiner. In any year, each medical examiner may require him to serve one month. The associate medical examiners in the other counties shall, in the absence of the medical examiners, or in case of their inability to perform their duties, perform, in their respective districts, all the duties of medical examiners.

Sec. 4. Each medical examiner and associate medical examiner shall, before entering upon the duties of his office, be sworn and give bond for the faithful performance thereof, in the sum of $5,000, to the treasurer of the county, with sureties to be approved by him. If he fails for thirty days after appointment to give such bond, his appointment shall be void.

Sec. 5. A surety on such bond, his heirs, executor, or administrator may apply to the superior court to be discharged from further liability thereon, whereupon the proceedings shall be the same as are prescribed in § 6 of chapter 23.

Sec. 6. Upon a breach of the condition of such bond to the injury of any person, the obligor may be removed from office, and actions may be brought upon such bonds in like manner as upon the bond of a sheriff.

Sec. 7. In the county of Suffolk, each medical examiner shall receive from the county an annual salary of $4,000, and the associate medical examiner, a salary of $666; but if the associate medical examiner serves in any year more than two months, he shall, for such additional service, be paid at the same rate, to be deducted from the salary of the medical examiner at whose request he serves. Medical examiners and associate medical examiners in other counties shall receive fees as follows: For a view without an autopsy, $5; for a view and autopsy, $30; and for travel, 10 cents a mile to and from the place of view.

Sec. 8. Medical examiners shall, in all cases, certify to the city or town clerk, or to the city registrar in the place where the deceased died, his name and residence if known, otherwise a description of such person as full as may be, with the cause and manner of his death, and shall make examination, upon a view of the dead bodies, of only such persons as are supposed to have come to their death by violence.

Sec. 9. If a medical examiner has notice that there is within his county the body of a person supposed to have come to his death by violence, he shall forthwith go to the place where such body lies, and take charge of the same; and if, on view thereof and personal inquiry into the cause and manner of death, he considers a further examination necessary, he shall, upon being thereto authorized in writing by the district attorney, mayor, or selectmen of the district, city, or town in which such body lies, make an autopsy in the presence of two or more discreet persons, whose attendance he may compel by subpœna. Before making such autopsy he shall call the attention of the witnesses to the position and appearance of the body. He shall then and there carefully record every fact and circumstance tending to show the condition of the body, and the cause and manner of death, with the names and addresses of said witnesses, which record he shall subscribe. If a medical examiner or associate medical examiner considers it necessary to have a physician present as a witness at an

autopsy, he shall be paid $5 for his services. Other witnesses, except officers named in § 42 of chapter 204, shall be allowed two dollars each.

Sec. 10. He shall forthwith file with the district attorney of his district a report of each autopsy and view of his personal inquiries, with a certificate that, in his judgment, the manner and cause of death could not be ascertained by view and inquiry, and that an autopsy was necessary. The district attorney, if satisfied that an autopsy was necessary, shall so certify to the county commissioners having jurisdiction in which the autopsy was held, or, in the county of Suffolk, to the auditor of Boston. If, upon such view, personal inquiry, or autopsy, the medical examiner is of opinion that the death was caused by violence, he shall at once notify the district attorney and a justice of the police, district, or municipal court, or trial justice having jurisdiction over the place in which the body was found, and shall file an attested copy of the record of his autopsy in such court, or with such justice, and with the district attorney.

Sec. 11. The court or trial justice shall thereupon hold an inquest, from which all persons not required by law to be present may be excluded, and the witnesses may be kept separate, so that they cannot converse with each other until they have been examined. The district attorney, or some person designated by him, may attend the inquest and examine the witnesses. An inquest shall be held in all cases of death by accident upon a railroad, and the court or justice holding such inquest, or an inquest in case of death by accident upon a street railway, shall give seasonable notice of the time and place thereof to the board of railroad commissioners. The attorney general or the district attorney may direct an inquest to be held in the case of death by casualty.

Sec. 12. If it appears that the place in which the body was found is without the limits of the judicial district of the court, or the territorial jurisdiction of the trial justice to which or to whom the notice required by the provisions of section ten has been given by the medical examiner, such court or trial justice shall nevertheless proceed with the inquest and have continuous and exclusive jurisdiction thereof, if said place is within the commonwealth, and within fifty rods of the nearest point in the boundary line of such district or territory, unless a similar but prior notice relative to the same body shall have been issued by a medical examiner for a county adjoining such district or territory at the point aforesaid.

Sec. 13. If an inquest is to be held by a police, district, or munici-

pal court, the court may appoint an officer, qualified to serve criminal process, to investigate the case and to summon the witnesses, and may allow him additional compensation therefor, which shall be paid in like manner as the fees of officers in criminal cases.

Sec. 14. If a magistrate has reason to believe that an inquest to be held by him relates to the death by accident of a passenger or employee upon a railroad, or of a traveler upon a public or private way at a railroad crossing, or to a death by accident connected with the operation of a street railway, he shall cause a verbatim report of the evidence to be made and sworn to by the person making it, and the report and the bill for services, after examination and approval in writing by such magistrate, shall be forwarded forthwith to the board of railroad commissioners. Such bill when approved by said board, shall be forwarded to the auditor of accounts and be paid by the commonwealth, assessed on the several corporations owning or operating the railroad or street railway on which the accident occurred, and shall be collected in the manner provided in § 10 of chapter 111. The magistrate may, in his discretion, refuse fees to witnesses in the employ of the company upon whose railroad the accident occurred.

Sec. 15. The magistrate shall report in writing, when, where, and by what means the person came to his death, his name, if known, and all material circumstances attending his death; and if it appears that the unlawful act of any person contributed to the death, he shall further state the name of such person, if known to him; and he shall file such report in the superior court in the county in which the inquest is held.

Sec. 16. If the magistrate finds that murder, manslaughter, or an assault has been committed, he may bind over, as in criminal prosecutions, such witnesses as he considers necessary, or as the district attorney may designate, to appear and testify at the court having jurisdiction over such crime.

Sec. 17. If a person charged by the report with the commission of a crime is not in custody, the magistrate shall forthwith issue process for his apprehension, returnable before any court or magistrate having jurisdiction of the case.

Sec. 18. The attorney general or the district attorney may, notwithstanding the medical examiner's report that a death was not caused by violence, direct an inquest to be held.

Sec. 19. The medical examiner may, if he considers it necessary, employ a chemist to aid in the examination of the body, or of substances supposed to have caused or contributed to the death, who

shall be entitled to such compensation as the medical examiner certifies to be just and reasonable. A clerk may be employed to reduce to writing the results of a medical examination or autopsy, and shall be allowed for his services $2 a day.

Sec. 20. No embalming fluid, or any substitute therefor, shall be injected into the body of any person supposed to have come to his death by violence, until a permit in writing, signed by the medical examiner, has first been obtained.

Sec. 21. After an autopsy, or a view, or examination without an autopsy, the medical examiner shall deliver the body, upon application, to the husband or wife, the next of kin, or to any friend of the deceased, who shall be entitled to priority in the order named. If the body is unidentified or unclaimed for forty-eight hours after the view thereof, the medical examiner shall deliver it to the overseers of the poor of the city or town in which it is found, who shall decently bury it, in accordance with the provisions of § 20 of chapter 81.

Sec. 22. Medical examiners, within their respective districts, shall, upon application and payment or tender of a fee of $5, view, and make personal inquiry concerning, a body intended for cremation.

Sec. 23. If services are rendered in bringing to land the body of a person found in any of the harbors, rivers, or waters of the commonwealth, the medical examiner may allow reasonable compensation therefor; but this provision shall not entitle any person to compensation for services rendered in searching for a dead body.

Sec. 24. The medical examiner shall take charge of any money or other personal property of the deceased, found upon or near the body, and deliver it to the person entitled to its custody or possession, or, if not claimed by him within sixty days, then to a public administrator.

Sec. 25. A medical examiner who fraudulently neglects or refuses to deliver such property to such person, within three days after demand upon him therefor, shall be punished by a fine of not more than $500, or by imprisonment for not more than two years.

Sec. 26. Every medical examiner shall return an account of the expenses of each view or autopsy, including his fees, to the county commissioners having jurisdiction over the place in which the examination or view is held, or, in the county of Suffolk, to the auditor of Boston, and shall annex to his return the written authority under which the autopsy was made. Such commissioners or auditor shall audit such accounts, and certify to the treasurer of the county what

items therein are just and reasonable, which shall be paid by him to the person entitled to receive the same. The commissioners shall not certify for payment any fee for an autopsy until the certificate of the district attorney required by the provisions of § 10 has been filed with them.

Sec. 27. Every medical examiner shall annually, on or before the first day of March, transmit to the secretary of the commonwealth certified copies of the records of all deaths, the cause of which he has investigated during the preceding calendar year, and, within sixty days after the expiration of his term of office, he shall make like returns for so much of the year as he was in office. For a refusal or neglect to make such returns he shall forfeit, not less than $10 nor more than $50.

Sec. 28. The fees of trial justices shall be as follows: For filing an attested copy of the record of an autopsy, 50 cents; for each subpœna issued, 10 cents; for each day's attendance in holding the inquest, $5; for the recognizance of witnesses, 20 cents; and for drawing up and filing a report in the superior court, $5. Special justices of police, district, or municipal courts, except those entitled to compensation other than that provided by § 69 of chapter 160, shall receive the same fees as trial justices. Said fees, having been audited by the district attorney, shall be paid by the county. Each medical examiner, including the medical examiners in the county of Suffolk, shall receive from the commonwealth 20 cents for each of the first twenty deaths recorded and returned by him in any year as above provided, and 10 cents for each additional death so recorded and returned, as certified by the secretary of the commonwealth.

Sec. 30. The secretary shall, at the expense of the commonwealth, prepare and furnish to the medical examiners blank record books, and blank forms for returns, and shall cause the returns for each year to be bound together in one volume with indexes, and prepare therefrom such tables as will render them of utility, and shall make report thereof annually to the general court in connection with the report required by § 22 of chapter 29.

848.—GENERAL STATUTES OF CONNECTICUT, REVISION OF 1902, TITLE 9, CHAPTER 3.

CORONERS.

Par. 1771. *Appointment of coroners.*—The judges of the superior court shall appoint for each county, upon the recommendation of the

state's attorney for such county, a coroner, who shall be attorney at law residing in such county, who shall hold his office for the term of three years from the time of his appointment and until another shall be duly appointed in his stead, unless he be sooner removed from the office by said judges, who may, for cause shown, so remove him, and who may, on the recommendation of the state's attorney for the county, fill any vacancy in said office of coroner.

Par. 1772. *Coroners shall be sworn; bond.*—The coroners for the several counties, before entering upon the duties of their respective offices, shall severally be sworn, and give bonds in the sum of $3,000 to the state, conditioned for the faithful performance of their duties.

Par. 1773. *Appointment of medical examiner.*—The coroner of every county shall, in writing, under his hand, appoint for each town in the county an able and discreet person, learned in medical science, to be medical examiner, who shall give a bond in the sum of $1,000 to the coroner for the faithful discharge of the duties of his office, and shall hold this office at the pleasure of the coroner. Each such appointment shall be recorded in the records of the superior court in the county where made; and, whenever any medical examiner shall be removed, the coroner shall deliver to him a written discharge, and file a copy thereof for record, with the clerk of the superior court.

Par. 1774. *Reports of violent deaths to medical examiner.*—When any person shall come to a sudden, violent, or untimely death, and when any person shall be found dead the manner of whose death is not known, anyone who shall become aware of such death shall forthwith report the same to the medical examiner for the town in which the dead body lies. Said examiner shall pay the person first reporting such death fifty cents therefor, and shall without delay repair to view and take charge of the body.

Par. 1775. *Returns by medical examiner.*—Whenever, after such view and immediate inquiry, the medical examiner shall be satisfied that the death was not caused by the criminal act, omission, or carelessness of another or others, and that there are no suspicious circumstances attending the same, he shall forthwith make out, sign, and leave with the registrar of births, marriages, and deaths of the town, a certificate of death in the form required by law, and he shall also, immediately after such view and inquiry, make out, and mail or deliver to the coroner of his county, a certificate signed by him of the following or similar import:

I, the undersigned, A. B., medical examiner, having notice of the death of C. D. (white or colored) ——, male, — years old, late of

the town of ———, in ——— (or, if unknown person, state that fact, and carefully describe the body, its clothing, and articles found on or near it which may be of service in its identification, always stating sex, color, apparent age, color and cut of hair, beard, color of eyes, and all special marks or deformities of body), —— who, on the ——day of———, A. D. 19—, (was found dead or died) in the town of ———, having viewed the body of said deceased, and made immediate inquiry concerning h— death, do hereby certify that said C. D. died in ———, on the —— day of ———, A. D., 19—, from (natural cause, suicide, or accident, as the case may be, stating manner of suicide and nature of accident), and that I am satisfied that the said death was not caused by the criminal act, omission, or carelessness of any other person or persons, and that an inquest is unnecessary. In accordance with the statute I have delivered the body of said deceased to (his friends or town authorities) for burial.

Dated, ———

A. B., Medical Examiner.

Par. 1776. *Medical examiner to notify coroner.*—Whenever a medical examiner shall see reason to suspect that the person whose body he has viewed came to his or her death by the criminal act, omission, or carelessness of another or others, he shall, as speedily as possible, by telegraph, telephone, or otherwise, notify the coroner for the county of such death, and of the place where the dead body is lying. Whenever the coroner has such notice, he shall at once, and on other notice may, proceed to view and take charge of the dead body, and make all proper inquiry respecting the cause and manner of the death; and, if from such view and inquiry he shall be satisfied that the death was not caused by the criminal act, omission, or carelessness of another or others, then said coroner shall make and sign the certificates required in like cases of medical examiners by par. 1775.

Par. 1777. *Autopsy.*—After a view and inquiry had by the coroner, if he shall have reason to suspect that the death was caused by the criminal act, omission, or carelessness of another or others, he may cause an examination or autopsy to be made of the body by the medical examiner, or by some other competent surgeon or physican, who shall reduce or cause to be reduced to writing, either at the time of making such examination or autopsy, or immediately thereafter, and, when practicable, in the presence of the body, every fact and circumstance found by such examination or autopsy which tend to

show the identity or condition of such body, and the time, manner, and cause of such death; which writing he shall subscribe under oath and deliver to said coroner. The taking of the testimony of such medical examiner, physician, or surgeon, and of any other person or persons whom the coroner may find it necessary to examine, shall constitute an inquest.

Par. 1778. *Coroner's jury.*—The coroner may, by warrant, cause a jury of six judicious men of his county to be summoned before him to assist him in his investigation, which jury shall be by him sworn to diligently inquire, under his direction, into the cause and manner of such death, and to present to him on their oaths and over their signatures a true verdict thereof. The coroner shall instruct said jurors in their duties, and as to all questions of law that may properly arise at such inquest; he may adjourn such inquest to meet again at such time and place as he shall think proper, and he may order any inquest or any part thereof to be held in private, in which case only the persons designated by him shall be allowed to remain in the room or place where such inquest is being held.

Par. 1779. *Prosecuting officer shall be notified.*—If the verdict or finding in any inquest charges any person or persons with having caused the death which is the subject of the inquest, the coroner shall, without delay, communicate the import of said verdict or finding to a grand juror or prosecuting attorney of the town or city in which such death happened or was caused.

Par. 1780. *Return of result of inquest.*—The coroner shall reduce to writing, and shall, within ten days after any inquest has been held, return the testimony of all witnesses examined in the inquest to the clerk of the superior court in his county, together with his report of the inquest, which shall include his finding or the verdict of the jury, and he shall also return to said clerk all certificates sent him by the medical examiner in accordance with par. 1775, and all similar certificates by him made.

Par. 1781. *Records shall be kept by coroner.*—Every coroner shall keep a proper and durable record book, into which he shall copy all the certificates sent to or made by him, as prescribed by par. 1775 and par. 1776, and in which he shall also keep a record of all views, inquiries, and inquests by him made or held, stating the time and place of the view and inquest, the name and residences of the witnesses, the cause and manner of the death as found, the disposition and place of burial of the dead body, and a careful description of all dead bodies not identified before burial.

Par. 1782. *Statement of person injured by another to be taken.*—Whenever a coroner has notice that there is in his county a person who has been dangerously wounded or injured by the criminal act, omission, or carelessness of another, and who is likely to die from such wounds or injuries, he shall endeavor to take, or cause to be taken, the statement of such person concerning the manner in which, and the person by whom, such injuries were inflicted.

Par. 1783. *Disposition of property found on deceased person.*—In all cases arising under the provisions of this chapter, the medical examiner shall take into possession, and shall deliver to the coroner, all the property found upon or near the deceased person, or which, in his judgment, will aid in the investigation of the death; and, when such articles are no longer required to be kept for the purposes of justice, the coroner shall deliver the same to the person or persons entitled to their custody, or, if they are not claimed by such persons within sixty days thereafter, then such articles or property shall be administered upon according to law.

Par. 1784. *Disposition of body.*—At the termination of all proceedings by the medical examiner and coroner, the body shall be forthwith delivered to the friends of the deceased for interment; but, in case there are no friends who shall take charge of and bury it, then to the proper authorities of the town in which such body is lying, whose duty it shall be to bury it. Whenever the deceased person shall not have left property sufficient to defray the expenses of the burial, then the same shall be paid by the said town.

Par. 1785. *Power of coroner in securing evidence.*—For the purpose of securing evidence, the coroner may enter any and all places in his county; shall have power to issue a warrant for a jury of inquest, to compel the attendance and testimony of witnesses by subpœna and capias issued by him or other proper authority, and to punish for contempt to the same extent as justices of the peace may in criminal cases; he may order and cause witnesses to be kept separate, so that they cannot communicate with one another until they shall have testified; he may order or cause any person or persons whom he has good reason to suspect of having criminally caused the death, in regard to which he is holding an inquest, to be arrested and committed to the county jail or other proper place; he shall have power to take bail in such cases, or commit without bail, when in his opinion the person arrested is guilty of a capital offense, and when the proof against such person is evident, or the presumption great. When bail is taken, it shall be to the state, and shall be conditioned for the

appearance of the person so arrested before the superior court for such county at its next criminal term. Any person so arrested and admitted to bail may be rearrested and committed by the coroner without bail, when, in the opinion of the coroner, the proof is evident, or the presumption great, that such person is guilty of a capital offense. In no case shall any person be kept under arrest by an order from a coroner for a longer time than twenty-four hours after the finding of the verdict by the jury, or after the finding of the coroner in such inquest; and, at any time before such finding, whenever the grounds on which a person is so arrested cease to exist in the opinion of the coroner, said coroner shall order the release of such arrested person.

Par. 1786. *Coroners may issue process.*—All process, orders, and papers, proper to be issued by a coroner, may be directed to any proper officer; and it shall be the duty of such officer to serve such papers and process as directed, and, when requested, to remain in attendance on such coroner, and to execute his lawful orders during any inquiry or inquest.

Par. 1787. *Penalties.*—Every officer who shall wilfully violate any provision of this chapter; and every person who shall wilfully and without good cause neglect or refuse to serve on a jury of inquest, when duly summoned; and every person who shall wilfully, and without good cause, neglect or refuse to report a case of death as prescribed in par. 1774 and par. 1789, or who shall wilfully and unnecessarily touch, remove, or disturb any dead body, or any particle on or near such body, or disturb its surroundings,—shall be fined not more than $500, or imprisoned not more than one year, or both.

Par. 1788. *Powers of deputy coroner.*—Every coroner shall, for such time as he may designate, appoint a deputy, who shall only act as such in case of the absence from his county, or inability of the coroner; and when so acting, such deputy coroner shall have the same powers and duties as are given to coroners; and while so acting, shall receive the same fees as the coroner in like cases.

Par. 1789. *Reports to medical examiners of another town.*—In case of absence or inability of the medical examiner, it shall be the duty of any person aware of the death of a person, under the circumstances named in par. 1774 to report the same forthwith to the most accessible medical examiner for another town, who shall thereupon perform the duties, and shall have the powers, of the local medical examiner.

Par. 1790. *Proceedings in absence of coroner.*—In case the at-

tendance of a coroner or of his deputy cannot be procured within thirty-six hours after the medical examiner has taken charge of a dead body, upon which an inquest ought to be held, or sooner, in case of the known inability of the coroner and his deputy to attend, the medical examiner who first took charge of such dead body may then hold such an inquest thereon; and in and for such case only, such medical examiner shall have all the powers and be subject to all the duties given to and prescribed for coroners by this chapter; but the coroner, or, in case of his inability, his deputy, may at any time enter and take control of such inquest, and thereupon such medical examiner shall be relieved from all further powers and duties therein.

Par. 1791. *Chemical or microscopical analysis.*—If in any case it shall appear to the coroner to be necessary to have a chemical or microscopical analysis, or other scientific investigation, for the purpose of ascertaining the cause of the death of the person on whose body he is holding an inquest, he shall so report to the state's attorney of his county, who may order such analysis or investigation to be made, and who shall certify to the expense thereof, which expense shall then be paid by the state on an order therefor by the clerk of the superior court of such county; the fees of any physician or surgeon for services rendered under the provisions of par. 1777 shall be certified and paid in the same manner.

Par. 1792. *Accounts of coroner.*—Every coroner shall return to the clerk of the superior court of his county a full account, signed and sworn by him, of all his lawful fees, expenses, and payments in each view or inquest, which account said clerk shall submit to the state's attorney for such county, who shall indorse the same, if correct, or such items thereof as are correct, and the sums so indorsed shall be paid by the state on an order thereof by such clerk.

CASES OF POISONING ILLUSTRATING IMPORTANT MEDICO-LEGAL QUESTIONS.

849.—Case I. Poisoning by Inhalation of Arsin During Smelting Silver and Lead Ore.

(Compiled by Assistant Professor Charles Harrington, M. D., of Harvard Medical School.)

(Vierteljahrsschrift für gericht. Med. und öffentl. Sanitätswesen, Bd. XVIII. p. 269.)

Dr. Trost, of Aachen, communicates a case of poisoning by arseniureted hydrogen, by which nine persons were affected, of whom

three died. At the lead works at Stolberg near Aachen, it was the custom to extract silver from lead ore by means of an old, expensive, and long process. Director Herbst, in Kall, formulated a new process for the extraction, which was as follows:

To the molten silver-containing ore is added 1½ per cent zinc; the mass is then heated to a higher temperature, since the melting point of zinc is higher than that of lead. The silver will by this means form an alloy with the zinc, and the alloy will appear on the surface on cooling. The alloy is then skimmed off and treated with hydrochloric acid, first cold, and then by moderate heat. The chlorin unites with the silver to form insoluble chlorid of silver, and with the zinc to form soluble chlorid of zinc, while the hydrogen is set free.

This process was carried out in September, 1869, at the lead works at Stolberg. On September 1st, at 8 A. M., several hundred weight of the alloy was treated in an iron kettle with several hundred weight of hydrochloric acid. The place in which the operation was carried on was a shed open on one side; in the roof were holes for ventilation. The mass in the kettle was stirred with an iron bar, and then heated. Although the formation of arseniureted hydrogen had been considered (since zinc and lead almost always contain arsenic), yet it had not been thought that the danger was so great as it afterwards proved. It may here be stated that, by the legal examination of the materials used, they were shown to contain large amounts of arsenic; the hydrochloric acid in particular contained 0.027 per centum. The operation was therefore performed in nothing less than a Marsh apparatus on a large scale, the gas evolved being diffused in the room. On the first day of the operation, towards noon, a number of workmen, who had stood near the kettle, were seized with symptoms of poisoning; the symptoms were, however, not much noticed. In the evening several others were seized, and on September 2d (the second day of the experiment) the operation had to be discontinued on account of the sickness of the workmen. The symptoms were the same in all the cases; and the treatment, which was carried out by several physicans, was without special influence on their course.

Of the six cases of recovery, one lasted but a day, one a little over two months, and four still longer. It will be enough to describe the course of those cases which ended in death.

1. Leonard M., thirty-seven year old, worked during both days, adding the hydrochloric acid and stirring the mass. Toward evening of the second day he was seized with a feeling of weight in the limbs, headache, abdominal pains, nausea, and vomiting. In the

night the feeling of weakness increased, the vomiting was more frequent, he had several liquid stools, bloody urine, delirium, and stupor. On the morning of September 3d he was found unconscious, with swollen face, the skin and the conjunctivæ colored yellow. The patient could be roused from the stupor for but a few seconds, and that with the greatest difficulty. The extremities were cold; the face covered with cold sweat; deglutition impossible; pulse small, 150 to 160. During the day he passed some bloody urine. Death occurred towards nine in the evening.

2. Th. S., forty years old, oversaw the operation from its beginning to its discontinuance. In the evening of September 2d he was seized with the same symptoms as Leonard M. On the morning of September 3d his condition was worse, and the abdominal pains were more violent. In addition, he had pain in the region of the kidneys, greater sense of weight in the limbs, dry and coated tongue, great thirst, but slight excretion of urine, and that bloody. The face was swollen; the skin of the entire body, and the conjunctivæ, were colored brownish-yellow; the head was hot. In the course of the day he had several brownish-black stools. As he passed no urine, the catheter was applied; but nothing except coagulated blood smelling of urine was obtained. The morning pulse was 110; the evening 130. On September 4th, pulse 130; body temperature 38½° C.; stools and urine as on the preceding day. September 5th and 6th, increased weakness; beginning delirium; restlessness; swollen abdomen; pulse 130 to 140; stools and urine as before. September 7th, sensorium dulled; stupor; face sunken; tongue and gums black; deglutition very difficult; pulse 160. Death occurred between three and four o'clock on the morning of September 8th.

3. Michael R., thirty-six years old, had worked at the kettle and was seized with the symptoms on the first day. The symptoms were like those of the two cases above. Death took place at 9 o'clock on the evening of Sept. 3d.

Autopsies were performed in all of the above cases and with the following results:—

1. Leonard M. Autopsy thirty-six hours after death.—Among the external appearances the following were worthy of note: Skin dirty yellow; about 30 grams of a blackish fluid smelling like garlic ran from the nose and mouth, on turning the corpse; the whites of the eyes were colored yellow and the pupils were widely dilated. The mucous membrane of the mouth and lips was colored dirty yellow.

The internal appearances of especial interest were as follows: The dura mater not injected; the pia mater much so. A slight layer of bloody serous fluid on the surface of the convolutions. The mucous membrane of the trachea, larynx, tongue, and mouth, was colored greenish-yellow; that of the œsophagus dirty-yellow. The liver was moderately large, and of a greenish-yellow color; the tissue of the same, hard and not rich in blood. Both kidneys were firm and congested; both colored dark-red. Bladder empty. The larger abdominal blood vessels filled with dirty-colored, dark blood. The tissues of the body, in general, were colored dirty yellow.

For chemical analysis were reserved the fluid from the nose and mouth, pieces of the lungs, heart, larynx, and trachea, liver, spleen, both kidneys, and the stomach.

2. Theodor S. Autopsy twenty-eight hours after death.—External appearances of especial interest were as follows: Skin—especially that of the face—was of a yellowish color; whites of the eyes dirty yellow; pupils half dilated. Lips and gums dirty blue. Abdomen somewhat swollen. Internal appearances: Pia mater injected; thin layer of serous fluid on the brain's surface. Auricles of the heart contained about an ounce of dark coagulated blood; the left ventricle was empty; the right contained a teaspoonful of coagulated blood. The pleural cavities contained about four ounces of dirty bloody fluid. The mucous membrane of the larynx and trachea was colored dirty yellowish-green, as well as that of the mouth and fauces. The mucous membrane of the œsophagus was dirty yellow. The stomach contained a spoonful of yellowish mucus. The liver was normal in size, firm, yellowish-brown in color, and not congested. The right kidney much congested, dark-red in color, normal in size; the left increased by a half in size, much congested, and of a brownish-black color. The bladder contained about a spoonful of bloody urine. There were reserved for chemical analysis, the fluid from the pleural cavities, the heart, portions of the lungs, larynx with trachea, portions of the liver and spleen, both kidneys, and the stomach.

3. Michael R. Autopsy sixty hours after death.—Among the external appearances were the following: The skin was bluish-green in color, and in places raised in blisters; whites of the eyes dirty yellow. A dirty dark-red fluid ran from the nose and mouth, on turning the corpse. The thorax, abdomen, and scrotum were distended. The internal appearances of interest were as follows: Dura mater not injected; pia mater much so; the surface of the brain covered with a thin layer of bloody serous fluid. The cerebrum, on re-

moval, broke down to a dirty yellow pap. In the pleural cavities was but a little dirty, dark, bloody fluid. The lungs were bluish-green in color. Heart empty. The great vessels contained a little dirty, dark-red blood. The mucous membrane of the larynx, trachea, mouth, and nose, was of a bluish-black color; that of the œsophagus was dirty yellow. The liver was enlarged, not very firm, contained not much blood, and was of a slate-blue color. The coats of the stomach dirty yellow, the stomach contained some fluid. The mucous membrane of the posterior wall of the same was, for an area of 2 square inches, colored black-gray, and was easily detached. The spleen was enlarged and congested; the tissue soft, and blackish-red in color. The kidneys normal in size, much congested, and of a dark-red color. Bladder empty. The large abdominal blood vessels contained some dark, dirty blood. The tissues of the body, generally, were of a dirty yellow color.

There were reserved for chemical analysis, the fluid from the nose and mouth, portions of the lungs, the heart, the larynx, and a portion of the trachea, part of the liver with the gall bladder, the spleen, kidneys, and stomach.

The portions reserved in each case were analyzed, and arsenic was discovered in all,—especially in the viscera.

From the above cases it will be seen that the symptoms of poisoning from respiration of the gas, and from internal administration of the arsenic, are very similar. And the post-mortem appearances of the two are also very similar, though, in the former, the local appearances are more prominent in the air passages, and, in the latter, in the stomach and intestines. In these cases the poison was introduced into the blood by the mouth, etc., and lungs. The gas in the body was resolved into arsenic and hydrogen; the arsenic was carried to the various parts of the body by the blood, upon which it, meanwhile, had its effect, and thus indirectly on the organs. The finding of the court in each of the above cases was that deceased came to his death by poisoning brought about by respiring arseniureted hydrogen.

850.—Case II. Poisoning by Arsenic in Coffee—Narcotic Form.

(Compiled by Assistant Professor Charles Harrington, M. D., of Harvard Medical School.)

(Maschka's Handbuch der gerichtl. Med. Vol. II. p. 260.)

Four persons, soon after drinking coffee in the afternoon of February 26th, were seized with abdominal pains and frequent vomiting.

Frau N. was as though unconscious, and could not move. Minna Z., a young girl six years of age, lay in a condition of supposed sleep; three hours after drinking the coffee she awoke, complained of pain in the abdomen, requested drink, took some sweetened water, and again fell asleep. On proceeding later to give her some medicine prescribed by the physician, she was found to be dead. Frau N. and her servant girl were for four days very ill; they vomited frequently. Herr N. was sick several days, but not confined to his bed. The autopsy of the child occurred on the day after its death. A foamy and somewhat bloody fluid ran from the nose and mouth; nothing abnormal about the mucous membrane of the mouth. The cavity of the abdomen contained about 60 grams of yellowish, clear fluid. The stomach gave off no unnatural odor; it was dilated with gas. The contents of the stomach and duodenum consisted of a grayish-yellow, faintly acid fluid containing a small amount of food and mucus. The mucous membrane was pale, the glands decidedly swollen; otherwise, nothing remarkable. The organs of the abdomen were generally anemic, but not abnormal. The lower part of the omentum was considerably injected, as was also the peritoneal coat of the small intestines. The small intestine contained a grayish-yellow slimy fluid. The mucous membrane was pale, the follicles swollen. The large intestine contained a yellow, thin material like thin pap. The large blood vessels of the abdomen contained a moderate amount of dark fluid blood. There were no remarkable appearances about the organs of the chest. The pia matter was richly injected, edematous, and easily detached. The sinuses contained fluid blood. There was nothing abnormal about the brain.

The chemical examination proved the presence of arsenic in the coffee, and in the organs of the young girl. It was found in the stomach and contents, intestines and contents, brain, lungs, heart, spleen, liver, and kidneys. There was no odor of decomposition from the stomach and intestines on the ninth day.

851.—Case III. Poisoning by Arseniate of Copper. (Vert de Mitis.)

(Compiled by Assistant Professor Charles Harrington, M. D., of Harvard Medical School.)

(Annales d'Hygiène Publique, Jan. 1880, p. 23.)

Leontine Puthomme, aged seventeen, took, on December 7, 1878, at 5 p. m., 50 grams of vert de mitis in water. Vomiting occurred

in half an hour and was assisted by the administration of an emetic. On the following day she was admitted to the Lariboisière Hospital. Vomiting was incessant; there were coldness of the extremities, prostration, and a small weak pulse. Epigastric pain very pronounced. Dejections diarrheic and yellow. She complained of dimness of vision; there was no characteristic eruption or paralysis. She died on the evening of the twelfth. Prostration steadily increased from the first day. There was no delirium until several hours before death. Vomiting and diarrhea had ceased since the previous evening.

The autopsy was performed on December 14th. The integrity of the mucous membrane of the digestive tract was quite unimpaired. There was a slight congestion of the pharynx, without any ulceration. The mucous membrane of the stomach was pale, and of perfectly normal appearance; the same is true of the mucous membrane of the intestines. The liver was yellow, and fatty degenerated. There were no well-marked lesions in the other organs. Chemical analysis of the organs, vomited matters, and urine, gave the following results, which are expressed in milligrams:

	Arsenic.	Copper.
1. Brain	2.40	1.81
2. Stomach and pancreas—		
in 42 grams	0.40	
in 47 grams		0.20
3. Liver	13.30	9.02
4. Heart	trace	0.51
5. Lungs—		
in 100 grams	0.70	0.47
6. Intestines—		
in 100 grams	0.50	0.30
7. Kidneys	1.50	0.76
8. Hair, 9 grams	0.10	0.00
9. Mammæ—		
in 60 grams	0.20	
in 64 grams		0.30
10. Muscles—		
in 40 grams	0.10	
in 55 grams		0.12
11. Urine, 69 c. c.	0.17	trace
12. Vomitus, 286 grams	0.28	0.25
13. Vomitus, 0.977 grams	1.41	0.79
14. Vomitus, 16.54 grams	2.494 grams	0.843
15. Vomitus, 2.409 grams	0.526 grams	0.316

Analysis of vert de mitis shows it to contain 22.64 per cent of arsenic, and 14.93 per cent of copper; a portion of the arsenic is

present as arseniate of sodium; 100 grams of the vert de mitis contains 4.82 grams of arsenic in this form. The girl ingested 50 grams of the substance which, by the above analysis, corresponds to 11.32 grams of arsenic, and 7.46 of copper.

852.—Case IV. Poisoning by Arsenic.

(Compiled by Assistant Professor Charles Harrington, M. D., of Harvard Medical School.)

(From the shorthand reports of the trial.)

Miss H., a woman of about thirty-eight years, strong and well, took from her brother, on May 1, 1879, at 10 P. M., a cachet of what purported to be soda, which he said was to settle her stomach, although there was nothing the matter with that organ. She soon went to bed, and, at about midnight, awoke in great distress. She complained of a "stopping of all her functions" and of a creeping sensation. At her request she was rubbed. The brother then brought her some "Jamaica ginger," which she drank. She then slept until between 5 and 6 A. M., when she awoke and complained of weakness and exhaustion. The brother then said he would give her some mustard; he mixed something, and gave it to her. Soon afterwards she called her aunt, complained of nausea, and asked for some mustard. This was given her, and she vomited. The brother, who was meanwhile away from the house, returned at noon, and carried her some tea. He then went to a physician, told him something of the case, and was ordered to give magnesia once in two hours. This he gave at 3 o'clock, in milk, and at five, in water. When preparing this he was each time alone in the pantry with the door shut. She grew worse and vomited. At 7 o'clock she received a third dose, and the vomiting continued. The brother then started leisurely for a physician. When seen by the latter, she was lying on her right side and back with the legs slightly flexed; she was quiet, pale, and somewhat bluish under the eyes. She had nausea, but no pain; the body was cool; pulse weak at right wrist, almost wanting at left. Hot bricks and mustard paste were ordered. Jamaica ginger and whisky were given; also subnitrate of bismuth for the nausea. The vomitus was about a pint of thick, slimy, greenish fluid. At midnight she was in no pain, and fully intelligent. Respiration was difficult and micturition impossible; she had several loose discharges. She was given a teaspoonful of whisky, which she swallowed; she could take no more. Soon afterwards she was dead, just before death she was unconscious and nearly pulseless; pupils dilated. The autopsy was performed by the medical examiner on the following day. There was no cadaveric

smell. There was a general blueness of the skin,—especially of the ears. There was some fluid on the brain surface. The lungs contained air and much dark bluish or black fluid blood. Heart normal. The stomach looked dark blue before removal; it was red or bluish-red at the upper part. The vessels of the stomach and intestines were unusually full. The mucous membrane of the stomach was reddened throughout, and marked in places by spots of a brighter red. The intestine was reddened for two or three feet. There were some particles of white powder on the gastric mucous membrane and some gritty particles in the intestines. The lower portion of the intestines was normal. The liver was normal in size, congested, and fatty degenerated. The kidneys were congested; one kidney had a congested patch under the capsule. Spleen normal. There was dark fluid blood everywhere. The heart was full, but contained no clots. The contents of the stomach were about eight ounces of a dark brown fluid.

The post-mortem appearances were, therefore, not inconsistent with the theory of arsenic poisoning. The absence of pain during the sickness, and the creeping sensation in the skin, were thought to have been the result of administration of morphin. It was known that the accused had arsenic and other chemicals in his possession, and that, on the day previous to the sickness, he had purchased 8 grains of morphin, of which but 6 remained. The organs of the deceased were submitted to Drs. E. S. Wood and H. B. Hills, of the Harvard Medical School, for chemical analysis. Arsenic was found in the following amounts:

In the stomach	5.78 grains.
In the intestine	3.86 grains.
In the liver	2.77 grains.
In the brain	a trace.
Total about	12.50 grains.

The prisoner's counsel made a very able plea, and, in the absence of conclusive proof, the accused was acquitted.

853.—Case V. Poisoning by Arsenic with Fatty Degeneration of the Liver, Kidneys, and Gastric Glands.

(Compiled by Assistant Professor Charles Harrington, M. D., of Harvard Medical School.)

(Transactions of the Massachusetts Medico-Legal Society, Vol. I. No. 1, p. 43.)

Harry C., aged four years, previously in good health, was taken, on the morning of November 19, 1877, with vomiting and gastric

distress. As the physician, who was called, failed to recognize the true nature of the attack, and no appropriate remedies were administered, the case may be said to have progressed without treatment. According to the statements of the child's attendants, the symptoms above mentioned continued with increasing severity until the morning of the 21st, about forty-four hours after the onset of the attack, when death occurred, preceded by stupor and convulsions. The vomited matters, which were dark-colored towards the last, were thrown away. Suspicions having been excited as to the cause of death, the medical examiner, Dr. Pinkham, was summoned to investigate the matter, and an autopsy was made thirty-four hours after death, Dr. Lovejoy assisting.

The body was found to be well nourished. It presented the usual appearances resulting from hypostatic congestion. There was no rigor mortis. A careful examination was made of all parts accessible without section, and nothing abnormal was discovered except slight parching of the lips, and some excoriation on the inside of the right cheek.

On section of the head, the dura mater was found firmly adherent to the calvaria, as is usually the case in young children. There was a moderate amount of serum in the lateral ventricles, but perhaps no more than normal. The brain and its membranes were, in appearance, healthy. On laying open the abdominal cavity, attention was immediately arrested by the large size and bright yellow color of the liver, the condition of which was obviously one of extreme fatty degeneration. On raising its lower margin, the gall bladder was seen to be distended with bile, some of which had transuded, staining the tissues beneath. The spleen was of normal size and color. The kidneys were of somewhat lighter color than usual, this appearance being especially observed in the cortical portion after section; but some of the pyramids of Malpighi were very dark. The stomach was removed entire, ligatures being placed above and below, and it, together with the liver, kidneys, and spleen, was sent to Dr. W. B. Hills, of the Harvard Medical School, for a chemical and microscopical examination.

The intestines were carefully examined, and it was found that the mucous membrane of the duodenum was intensely red and swollen, and covered with a tenacious coating of mucus. These conditions obtained also in the jejunum and ileum, decreasing from above downwards. The mucous membrane of the colon was somewhat reddened. The rectum was partially distended with solid feces, its mucous mem-

brane being normal in appearance, or nearly so. The bladder was empty and contracted. The heart and large blood vessels appeared healthy. The right auricle was distended with black, somewhat firmly clotted, blood; the left auricle was empty. The right ventricle contained a small amount of blood, partially clotted. The left ventricle contained a small clot, a portion of it decolorized. The valves were normal. The larynx, trachea, bronchi, lungs, and pleuræ were healthy in appearance. The spinal cord was carefully examined, and no indications of disease or injury were discovered in either it or its membranes.

Dr. Hills returned the following report, which is presented in a somewhat condensed form: The stomach contained 2½ ounces of a dark brown viscid liquid, which, on standing, deposited a reddish-brown sediment containing many dark red bits of substance of varying size, which, on microscopic examination, proved to be little clots of blood. The color of the liquid was found to be due to admixed blood and bile. In addition to blood corpuscles, the microscope showed epithelial cells from the glands of the stomach, a large amount of granular matter, and a considerable number of crystals of arsenious acid. In the course of four or five hours' searching, perhaps three hundred were discovered.

The mucous membrane of the stomach was observed to be somewhat swollen and covered with a closely-adhering coat of mucus. It was for the most part pale in color, but reddened in patches, particularly over the greater curvature, the redness assuming principally a clotted form. There were also a few small dark patches near the pylorus, due to blood effused beneath the mucous membrane. Under the microscope, the liver appeared to be in a state of extreme fatty degeneration, a fact obvious, indeed, to the unaided eye. There was quite extensive fatty degeneration of the kidneys, and also of epithelium lining the peptic glands. One fifth of the contents of the stomach, and one fifth each of the liver, kidneys, and spleen, were tested together for the mineral poisons by appropriate methods. The result of this analysis was the discovery of arsenic. A portion of the liver (one fifth) tested separately for arsenic failed to show its presence. The contents of the stomach, tested as in the first instance, revealed unmistakably the presence of arsenic. No separate examination was made of the spleen or kidneys. It was not thought advisable to make a quantitative analysis, without a special order from the medical examiner or court. It was estimated, however, that there might be ½ grain of white arsenic in the contents of the stomach.

854.—Case VI. Poisoning by Arsenic.

(Taken from text of a former edition.)

We are indebted to Prof. George F. Barker, professor of physiological chemistry and toxicology at Yale College, for the following testimony, which he gave in the case of Horatio N. Sherman, poisoned with arsenic by his wife. As the case has never yet appeared in any work on toxicology, as Mrs. Sherman was convicted mainly by the evidence of the chemical and medical experts, and as we have a copy of the chemical evidence corrected by the professor himself, we make no apology for introducing verbatim in this work the evidence of the chemical expert as given in court.

After certifying to the identity of the stomach which had been committed to his hands by Dr. Pinney, Prof. Barker describes the chemical process by which he assured himself of the presence of arsenic in the viscera from Mr. Sherman's body:—

The stomach was ligatured at both orifices; these ligatures I removed. I then opened the stomach by making an incision along the lesser curvature; I emptied the contents into a clean dish and proceeded to wash the inner surface of the stomach with distilled water for the purpose of ascertaining its condition; I found it to be considerably congested throughout, there being patches showing distinct inflammation about the cardiac orifice and at the pylorus. The inner surface was nowhere softened or ulcerated. The contents, which were poured out, consisted of 3 or 4 ounces of a dark, offensive, slimy mucus, containing, apparently, bile. The second package in the box, on taking off the paper, was found to be a roll of new cloth, containing a portion of a human liver. This presented no unusual appearance, being perfectly healthy, with the exception of a small cyst on one side; after making notes of these results, I carried the materials from my laboratory to that of the medical college, and placed the liver in a new, clean glass jar, under double lock and key. I should here mention that in this package was a fragment of a large intestine, the mucous surface of which appeared slightly inflamed. The next day, two thirds of the stomach, and the contents of the stomach, were taken by me from the jar for the purpose of examination. At the conclusion of the examination of these organs, a portion of the liver was likewise examined. The result of the examination was the finding of arsenic in both these organs. On the third of June, I reported to the grand jury of Derby the presence of arsenic. Subsequently, I examined the remaining third of

of the stomach and a weighed portion of the liver (6⅔ oz.), for the purpose of obtaining the quantity of arsenic present in that organ. The method of analysis followed in all these cases was as follows:—

The portion of the organ to be examined was finely divided, placed in twice its bulk of distilled water, strongly acidulated with pure hydrochloric acid, a few grams of potassium chlorate were added from time to time, and the whole gently heated. In the course of eight or ten hours the mass was dissolved, and a clear yellow liquid was obtained. This was filtered, heated till the odor of chlorin disappeared, and then a washed stream of hydrogen sulphid gas was passed through it for from twelve to sixteen hours. The liquid was again filtered, and the precipitate thus obtained collected upon a filter, washed till free from chlorin, dried on the water bath, oxidized with pure nitric acid, and then heated with pure sulphuric acid, the heating being continued till the mass was completely charred. Water was then added and a little pure hydrochloric acid, and the whole was gently heated and filtered and the residue washed. Through the filtrate and washings, hydrogen sulphid gas was again passed for twelve hours. The precipitate thus produced was filtered off and washed, and upon it was poured upon the filter some pure ammonium hydrate and sulphydrate, previously heated to boiling. The filtrate was received in a porcelain capsule, evaporated to dryness with a gentle heat, and the residue oxidized by repeated evaporations with pure nitric acid; pure sodium carbonate and nitrate were then added, the whole dried carefully and heated to complete fusion. After cooling the fused mass was dissolved in cold water, the solution filtered, pure sulphuric acid was added, and it was evaporated till the fumes of the acid appeared. To it was then added a saturated solution of sulphurous acid, and it was again evaporated until fumes appeared. It was then diluted with water, and through it was passed washed hydrogen sulphid gas; a heavy, yellow precipitate of arsenious sulphid was thus obtained. This was filtered off, washed, and dissolved in pure ammonium hydrate poured on the filter. The filtrate was received in a previously weighed porcelain capsule, and evaporated to dryness on the water bath; the capsule was then reweighed, and the increase in weight showed the amount of arsenious sulphid which had been added; treated in this way, the 6⅔ ounces of liver examined in the second case yielded .602 of a grain of sulphid of arsenic, which quantity I now produce. [The professor exhibited a small vial containing the article.] This quantity corresponds, by calculation, to .485 of a grain of white arsenic,

or ½ grain. Calling the weight of the liver 4 pounds, which appears to me to be a fair average,—the quantity obtained in two separate and distinct examinations of the same liver being the same relatively to the size taken in the two cases,—my opinion is that this entire liver contained nearly 5 grains of white arsenic.

Prof. Barker resumed: The sulphid of arsenic produced in the bottle was from a weighed portion of the liver; another portion of sulphid of arsenic was obtained from an unweighed portion; this was oxidized with pure nitric acid by repeated evaporations; the excess of acid was then removed by evaporation with distilled water; the residue was dissolved in distilled water; a small portion was removed and tested with ammonio-silver nitrate; on the addition of this reagent, a brick-red precipitate of silver arsenate was produced. This was inclosed in a small tube, and is contained in the tube herewith produced. [A framed card was here shown, with this tube among others placed on it, with its contents, and marked "silver arsenate."] The rest of the solution was evaporated with a saturated solution of pure sulphurous acid in water, until all odor disappeared; two portions of the liquid thus obtained were removed; one of them, on being tested with ammonio-nitrate of silver, gave a canary-yellow precipitate of silver arsenite, which rapidly became dark olive-brown on exposure to light; this precipitate is here produced, marked "silver arsenite;" the second small portion taken was tested with ammonio-cupric sulphate; a light green precipitate of copper arsenite, or Scheele's green, was thus produced; this is here marked "copper arsenite." The remaining portion of the liquid was divided into two equal parts. One of these (A) was evaporated nearly to dryness, mixed with sodium carbonate in excess, and thoroughly dried. The dried mass was then intermixed with a perfectly dried mixture, composed of 1 part of pure potassium cyanid and 3 parts of sodium carbonate. Several portions of this mixture were then heated in hard glass tubes, having a bulb blown on one end. Each of these gave a black, lustrous, mirror-like deposit of metallic arsenic. One of these tubes is here produced, marked "metallic arsenic by the reduction test." A second similar tube was prepared, sealed, and the portion containing the arsenic heated; the deposit was readily volatile and condensed in the cooler portions of the tube in a brilliant crystalline sublimate, consisting of distinct octahedral crystals of arsenious oxid or white arsenic. This tube is here produced. [A magnifying glass was produced, and the crystals distinguished by the witness.] Other portions of the dry mass were

then tested by heating them in a stream of carbonic gas. (Fresenius and Babo's test.) Each of the portions thus heated gave a bright mirror of metallic arsenic, similar to that obtained in the reduction test. One of these tubes is here produced. No other substance but arsenic gives this test. A second tube was heated, the deposit volatilized, and a ring of crystals of white arsenic was obtained as before. The tube containing these crystals, with some of the metal not oxidized, is here produced. The other half (B) of the liquid was divided into two portions; one of these was tested by the method of Reinsch; *i. e.*, it was acidulated with hydrochloric acid, and in it strips of copper were boiled. These strips became covered with a steel-gray arsenical coating, which finally became black. One of these strips is here produced, marked "metallic arsenic on copper —Reinsch's test."Another copper strip was heated in a small tube, and afforded a sublimate of white crystals of arsenious oxid, distinctly octahedral by the microscope. These crystals I here produce, marked "arsenious oxid by Reinsch's test."

The second half of this portion was tested by the method of Marsh; it was added to a flask from which pure hydrogen was being evolved by the action of pure sulphuric acid upon pure zinc; the gas, as evolved, was freed from acid vapors and from moisture by passing it over solid potassium hydrate and calcium chlorid; it was then passed through a long tube of hard glass drawn out at intervals. Before adding the solution, the purity of the materials had been tested by heating this tube to redness, and allowing the gas to pass for an hour; no deposit was obtained in the tube. After adding a portion of liquid to be tested, a hair-brown deposit of metallic arsenic appeared in the narrow part of the tube just beyond the heated portion, deepening in color until it finally became black; two of these constricted portions of the tube containing the black deposit of metallic arsenic are here produced, marked "metallic arsenic, in tube, by Marsh's test." At the same time the flame of the burning hydrogen at the end of the tube became bluish-white, and deposited upon a piece of cold porcelain held in it brown, mirror-like spots of metallic arsenic; two of these pieces of porcelain containing the spots are here produced. The other deposits in the constricted portions of the tube were then tested; the deposit was found to be readily volatile and oxidizable, giving a ring of crystals of white arsenic; this tube is here produced, marked "arsenious oxid, Marsh's test." Other portions were submitted to the action of hydrogen sulphid gas passed through the tube. On gently heating it, the black deposit of metallic

arsenic changed to the bright yellow of arsenious sulphid, or yellow arsenic. Two specimens of the deposits thus obtained are exhibited. The tubes contain a portion of the black metal, and beyond this a portion of the yellow produced from it. The same tests in the same order were applied to the portion of arsenious sulphid obtained from the contents of the stomach, and a series of products, obtained in the same way, are here exhibited at the top of the card, marked as before, in the same order on the card. During several of these tests, whenever the metallic arsenic vapor escaped into the air, the peculiar odor of garlic was observed. Besides arsenic, I obtained, both from the contents of the stomach and from the liver, sulphid of bismuth; that sulphid was converted into subnitrate, and a portion of it from each organ is here exhibited; the tubes are marked "bismuth subnitrate." All these tests throughout were made in duplicate. In order to ascertain whether the materials employed in this investigation were pure, I took, before the second examination of the portions of the body mentioned, a fragment of beef's liver, equal in size to the fragments of human liver previously taken, and examined it in the same way, using the same materials and the same apparatus, except where it was replaced by new articles. No trace of arsenic was obtained in this examination. My opinion, therefore, is, that the material and vessels employed were pure and clean. My opinion, also, is, that the yellow precipitate obtained from the weighed portion of the same human liver is also sulphid of arsenic.

To Col. Wooster.—Did not attempt to weigh the arsenic in the stomach as I did in the liver: arsenic, in chemical language, refers to a metal which is black and lustrous, and may be combined with oxygen or sulphur. When combined with oxygen, the form known as oxid is produced; it bears the same relation to arsenic that iron rust does to iron. [Here followed an explanation of the method in which metallic arsenic is obtained, for the enlightenment of the jury.] It is a question whether metallic arsenic is poisonous. It is a general principle that no metal, in itself, can be poisonous. The sulphid of arsenic and white arsenic are both poisonous, though the white arsenic is more so than the yellow.

Q. Have you any means of telling what quantity of arsenic there was, approximately, in the stomach you examined ?

A. The tissue of the stomach contained no arsenic; the contents of the stomach contained an amount which, though I did not weigh it, I estimated—[Objected to this as a mere guess. Objection overruled, the court holding that the professor ought to know.] It is

fair to assume that comparison by bulk is correct. Judging from the bulk obtained from the stomach, the quantity present must have been about one tenth of a grain.

Q. How long after arsenic is taken does it ordinarily find its way to the liver ?

A. White arsenic taken into the stomach is removed therefrom by absorption, and, as it has not been detected in either the lymph or the chyle, this absorption must take place by the blood. It has been detected in the urine within one hour after it has been taken. The liver acquires its maximum quantity, in the opinion of several authorities, in from fifteen to eighteen hours after administration.

Q. Does it pass off or remain in the liver ?

A. It is eliminated from the liver, and may entirely disappear in from eight to fifteen days after being taken; depending upon the quantity and other circumstances.

Q. What is the maximum quantity the liver of an ordinary adult will contain?

A. I think that the maximum quantity the human liver may contain is the quantity I have found in the present case.

Q. From the presence of this quantity of arsenic, have you an opinion as to how long it must have been taken before death?

A. At least fifteen hours; how much longer I cannot say.

Absorption and elimination of arsenic go on together; if there were vomiting and purging, a portion of the arsenic taken Monday night would have been removed before death on Friday. A considerable portion may have been eliminated, during the five days supposed, by the kidneys. Moreover, the arsenic is distributed through the entire body, and the analysis of a single organ or two does not discover the arsenic elsewhere contained. For both these reasons, the amount obtained cannot have been the whole amount taken, and must be less.

Q. Is arsenic a cumulative poison?

A. The question of cumulation depends upon the definition of the word cumulative. If by cumulative is meant the accumulation of a substance in the body, when it is taken more rapidly than it can be eliminated, then arsenic or apple pie may be regarded as cumulative. If by cumulative be meant a substance which enters into combination directly with certain tissues, so as to form a compound less readily removed than the original substance taken, or not removed at all, then arsenic in my opinion is not a cumulative poison. I have heard the testimony of Dr. Beardsley in relation to the sick-

ness of this man; the quantity of arsenic found in his stomach may have been, in my opinion, either the last traces of a very large quantity taken on Monday night, or the residue of a smaller quantity taken subsequently; which I cannot tell, but the latter appears to me the more probable. The first effect of arsenic when administered is local. It is specified as an irritant, but not a corrosive poison, that is, it inflames the parts it comes in contact with, without destroying, softening, or perforating them; the local effects thus first produced cause local symptoms; these appear generally in an hour after the poison has been swallowed. They are burning pain in the stomach, which, as the poison passes down, extends along the intestinal tract. It increases in severity, is accompanied by great thirst, dryness and constriction of the throat, vomiting and purging. The matters vomited are dark, bilious, and offensive. By this time more or less of the poison has become absorbed; it enters the blood and produces a second class of symptoms called "remote," the action being apparently mainly upon the blood corpuscles; these symptoms are characterized by great prostration of strength, anxiety and depression of mind, a peculiar lividity of the face, a blue line under the eyes, the intellect being as yet unaffected, and the mind clear; death may ensue at this stage of the action, due to the prostration usually taking place under these circumstances, in from one to eight days; if life be continued, the effect of the arsenic is apparent upon the nervous system and brain; stupor, passing into profound coma, may develop itself in case the action is primarily upon the brain; delirium and convulsions, passing into tonic spasms, in case the spinal cord is also involved, may precede death; all of these symptoms may not be present in any one individual; the patient may recover from all these symptoms, which may be called primary, and may die from the secondary effects of the poison years afterwards. White arsenic, when taken into the stomach, is dissolved, both in the stomach and intestines, by the gastric and intestinal juices; spread out upon the posterior surface of these intestines, is a set of blood vessels which compose what is known as the portal system; the arsenic thus dissolved passes into these blood vessels through their walls; these vessels unite, thus forming the large trunk known as the portal vein, which empties itself directly into the liver, so that this organ is not only more fully supplied with blood than any other, but is supplied with blood charged with arsenic from the intestines; hence the liver is more likely to contain this poison than any other organ. It passes from the liver into the general circulation, and thus produces its

remote effects. Most probably it does its fatal work on the blood itself, disintegrating the blood corpuscles, thus rendering the blood unfit to perform its functions. From 2½ to 3 grains of arsenic would kill an adult; less quantities than that have been fatal; and more has been taken without producing death. Arsenic would not probably be found if death ensued from the secondary effects. Persons have died from the primary effects of arsenic in eight days, and no trace of the poison has been found in the body on analysis. Constriction of the throat, when arsenic is taken, occurs from one half hour to an hour, ordinarily.

Q. Assuming that the liver you examined was Horatio N. Sherman's, of what do you think he died?

[Objected to, and admitted as the witness's opinion, based on his own analysis.]

A. My opinion is, that, considering the symptoms as described and in the presence of arsenic as stated, Horatio N. Sherman died from the effects of a poisonous dose of arsenic.

I found in a locked drawer, upon my return from New York in July, a package, which I now produce; a white paper, tied with a red string, with the word "bismuth" written on it, which I found to contain subnitrate of bismuth, upon opening it. Forty grains of it were examined for arsenic, with a negative result. The bismuth was pure.

The white crystals of white arsenic, not appearing distinctly in the tubes on the card, I have placed beneath them black paper to make them distinctly visible. I did not mention the effect of the poison on the action of the heart and lungs; the pulse, during great prostration, is generally small and rapid; sometimes, when the prostration is great, the pulse is less rapid than usual; the respiration is difficult, requiring almost a voluntary effort to breathe; it is sometimes quick and sometimes slow; the action of the poison on the lower intestines is to produce tenesmus, *i. e.,* straining without result; inflammatory action produces a dry and hot skin, at the first stages; after collapse, the skin is cold and clammy, and sometimes moist; the inflammation causes dryness and a folding up of the mucous membrane; there is also more or less faintness; sometimes entire syncope; purging may be wanting; convulsions are exceptional; the same is true of delirium.

Cross-examined by Mr. Watrous: I commenced the analysis on the 6th of November, 1871, and obtained the sulphid on the 23d of the same month; took the 6⅔ ounces from a portion of the liver left

at my office; first received it on May 13th; from that time until November 6th it was in my laboratory hidden behind some other bottles, in a sealed glass jar, in a cupboard which was locked; the laboratory was locked; only myself and the janitor had keys; was not in the laboratory daily; have no recollection of anyone being present while I was there; opened the bottle only once from May 13th to the 30th of June; it was not labeled; no one was present when I opened it; opened it in the daytime; gave the janitor instructions in regard to it; did not point it out to him; I pointed to the door, and said that that was not to be opened by him or anyone else by his knowledge; told him I had an important investigation in progress, and it mustn't be interfered with; didn't tell him what I was looking for; was looking for poison, not for arsenic. I was told that arsenic was suspected; was not requested to examine it in order to see if arsenic was there; looked for all metallic poisons; could have discovered antimony as readily as I did arsenic; was not told there was a suspicion of anything but arsenic; did not start in my investigation with that in view. Never made any experiment with the view of finding arsenic.

[The witness here appealed to the court to make an explanation, in reply to questions asked him; and the court held that the professor had a right to make any explanation to make his answers clear.]

Q. If you did not find arsenic either designedly or by accident, how did you find it?

A. I was looking for metallic poisons, and found arsenic. [The witness was going on to make an explanation, when Mr. Watrous objected, but finally withdrew his objection.]

The explanation: The examination is for metallic poisons, and not for one or two or three, but all. And if one is found, it is found without especially looking for it. If arsenic had been there alone, some other process than the one I followed might have been better. I looked at the jar, occasionally, to see that it had not been disturbed; don't know how many kinds of chemicals I mixed with the human liver during my analysis; think it was not fifty nor twenty-five, nor fifteen; possibly ten; cannot tell exactly, without counting.

[Mr. Watrous asked witness to count them up.]

Witness: If I have counted correctly, I used ten chemicals in the analysis; no means of getting at the bulk of the chemicals used. The following is a list of chemicals I used in the analysis: Water, hydrochloric acid, potassium chlorate, hydrogen sulphid, nitric acid, sulphuric acid, ammonia, ammonium sulphydrate, sodium nitrate,

sodium carbonate, and sulphurous acid. Tested the sulphuric acid and found it pure; sulphuric acid does sometimes contain arsenic; this is not true of sulphurous acid. I used ½ fluidounce, about 100 grains of sulphuric acid during the tests; know what I obtained was sulphid of arsenic.

It is not settled among chemists whether arsenic is a metal; it is not absolutely known that it is not itself a compound; the elements of it, if it is a compound, are of course not known; know that the process through which I put the liver would not produce arsenic; I have gone through the same process many times, and never generated arsenic; I know as well as I know anything, that arsenic was not generated in this case; I believe it as much as I believe I exist; I cannot prove that I exist; it is not claimed that chemists can manufacture diamonds or things that cannot be told from diamonds; have heard of the Wharton trial; have read the testimony of chemists claiming to be such, whose testimony differed as to the existence of antimony; the main difference was whether it was enough to cause death; not about the existence of it; arsenic in bismuth is very uncommon now; it is found sometimes with bismuth; if bismuth in which arsenic was put had been placed in the stomach, I should have found them separately; bismuth is not a poison; it would not kill a man if taken into his stomach; subnitrate of bismuth won't kill.

855.—Case VII. The Trial of Mrs. Mary L. Manson before Judge Milton A. Shumway, in the Superior Court, March 10, 1903, at Putnam, Connecticut, for the Murder of Mrs. Julia A. Wilson, who died at Ashford on the 6th day of July, 1902.

(Taken from court stenographer's report of said trial, a copy of which was furnished the editors.)

Arsenic was the supposed poison administered in this case. This case is somewhat confusing because there was evidence presented to show that Mrs. Wilson was in the habit of taking at odd times, without the advice of an attending physician, paregoric and morphin in tablets, swamp root, bromo-quinin with morphin in white powder, and arsenic. The first evidence of sickness occurred after a return from a visit at a friend's house. She complained of nausea, and a prickling sensation in her hands, and a swelling of her limbs. A daughter testified that her mother was never strong; this daughter (who was the person accused of poisoning her mother) also testified that "mother was not well since sister died, September, 1901."

Dr. Rienzi Robinson, of Danielson, performed the autopsy in the presence of four other physicians at a house on December 17th; he took the stomach (tying it with two ligatures both above and below, and cutting between the ligatures so that none of the contents could escape) which was not opened. This witness also took from the body a piece of the lung, the kidneys, the spleen, and a piece of the liver. These parts were given to Coroner Bill, who sealed them up in jars which had been sterilized. A mixture of alcohol and water was also placed in the jars. The witness held the stomach up to the sunlight and looked through it. He noted considerable inflammation. On December 21st, the witness said he made another examination of the body. It was disinterred and taken to a house about half a mile below the cemetery. The witness took from the body the brain entire; also pieces of the lung, liver, œsophagus, and duodenum; also the bladder, uterus, and a piece of the muscle of the right leg. There was no urine in the bladder. Coroner Bill took possession of these parts which were put in separate glass jars.

On December 24th, Dr. Robinson said he performed another autopsy at the graveyard. The casket was opened; but the body was not removed. The witness on this occasion took out what was left of the liver, and the tibia of the right leg. He did not detect any evidence of decomposition. Dr. Neff called the witness's attention to the mouth. He found the lips and mucous membrane of the mouth inflamed. The whole body had the appearance of a body that had been embalmed.

Coroner Bill testified that the jars above referred to were delivered to Dr. Wolff, of Hartford.

Dr. A. J. Wolff testified that he had received these jars from Coroner Bill and was informed by him that arsenical poisoning was suspected. He described fully the appearance of the jars and what they contained, and then went into a minute description of his microscopical examination, exhibiting to the jury pictures which he had taken of the slides prepared. There was no odor of decomposition about the stomach; the tissue had a fresh, pinkish look, and was extremely pliable. Inflammation of the pancreas and kidneys was apparent, and a fatty degeneration of the latter. The spleen, liver, and piece of the lung were for the most part normal, as was also the heart and brain. On examination of the bladder and kidneys, Bright's disease was very apparent. An examination of the liver revealed a fatty change due to some acute disease or irritant poison. The witness described in great detail his examination of the stomach. Acute in-

flammation was very apparent. Witness described to the jury the various tests to which he subjected the stomach and its contents, exhibiting glass tubes showing arsenical mirrors recovered from the tissues of the viscera by means of the Marsh test, which clearly showed the presence of large quantities of arsenic; also the copper foil used in another test, which was coated with arsenic. The witness showed the jury a number of photomicrographs of the diseased kidneys of Mrs. Wilson, also of arsenic crystals isolated from her viscera and stomach contents; some of the plates showed the octahedral crystals characteristic of arsenic, and positive proof of its presence.

The witness said he also made a quantitative analysis of the organs sent him, and explained clearly and definitely his methods, the results of the analysis being as follows:

from	the bladder and uterus	13.8	milligrams.
"	the stomach	30.	"
"	muscle, calf of leg (estimated)	.5	"
"	brain (estimated)	.1	"
"	the heart	7.1	"
"	the pancreas	15.2	"
"	the lungs	1.7	"
"	the large kidney	15.8	"
"	portion of liver used by witness	2.6	"
"	the duodenum	22.6	"
"	the stomach contents	133.6	"
"	the spleen	12.4	"

The total recovered from all of the tissues examined was 2.3 grains of arsenic.

Witness said that in his opinion Mrs. Wilson, having chronic renal disease, died from acute gastritis, inflammation of the liver, stomach, and kidneys, due to the presence of arsenic in her system. Arsenical poisoning was the cause of her death. In the witness's opinion poisoning had been going on some time, as arsenic was found in the bone examined. The witness said the presence of arsenic in the tissues shows that it caused her death. The arsenic in her stomach proves that it was given shortly before her death. The reflex action in the stomach was destroyed so that Mrs. Wilson could not vomit; and the action of the kidneys was suppressed so that large amounts of the arsenic had accumulated in the liver and kidneys.

Cross-examined by Attorney Torrey for the defense, the witness said that arsenic was used commonly in medicine. It was a very good tonic. Witness said he had heard of arsenic eaters in Connecticut. Arsenic produces, on sublimation, an octahedral crys-

tal, the only substance known that produces such a crystal. Where the kidneys were diseased, the arsenic would not readily be thrown off. Arsenic in this case might have been administered months before the death of Mrs. Wilson. The ordinary dose of arsenic at first is one or two drops (Fowler's solution) taken three times a day.

Witness said he had heard of 1 grain a day being given. He had practised medicine twenty-seven years, but had never heard of a person buying 1 ounce of arsenic to prepare for himself to take. The witness had received a white powder, some liniment, and a bottle marked "alcohol" from the coroner, and had tasted these for arsenic, but had found none. Witness said he knew that the crystals found embedded in the stomach walls were arsenious acid crystals. The crystals of salt are not octahedral in form. Proportionally, there was an unusual amount of arsenic in the spleen, liver, and pancreas.

The witness testified that the acute gastritis would have caused death in itself. He showed the distinction between acute and chronic arsenical poisoning, and explained in detail the symptoms in each case. The witness thought Dr. Converse was justified in his conclusion in regard to Mrs. Wilson's case. He showed the jury a colored plate of a stomach, which he said illustrated the color of the stomach which he had examined. It was the witness's opinion that two doses of arsenic were given to Mrs. Wilson; that one was fatal, and the other was not expelled from the stomach.

Another witness (George C. Wilson, a son) testified that he knew his mother took morphin. He had purchased it for her on three occasions, and his sister used to get it for her. Mrs. Wilson also took paregoric and laudanum. The witness had purchased a quart of paregoric for her at one time. On one occasion Mrs. Wilson had given the witness a slip of paper to take to the druggist. The witness said he had read what was written on the paper, and that it was a request for the druggist to give bearer fifteen or twenty cents' worth of arsenic. (Attorney Torrey here read the witness's statement made to Coroner Bill at the Brooklyn jail. In that statement the witness first said he had read this slip of paper given him by his mother; but thinking, as he said, that such a statement would get him into trouble, he asked the coroner if he might change this and say that he did not read the note.) The truth, he averred, was that he read the note. He procured the poison at Burt's drug store in Putnam. A red poison label was put on the package. The arsenic was put up in a round box, slightly larger than a pill box, and the witness had seen this box subsequently on the shelf, with other medicines his mother used.

He had often seen his mother take morphin and arsenic. She dipped a black pin, which she had moistened, in the powder, and then put it in her mouth. A very little would stick to the pin. Another witness, Mrs. Fenimore, of Eastford, testified that she went to the house of Mrs. Wilson for the purpose of taking care of her on October 8th, and remained there four days. When the witness arrived at the Wilson house, Mrs. Wilson was lying on the lounge. Her feet and limbs were much swollen, and her mouth and eyes were sore. Witness said she saw Mrs. Manson give Mrs. Wilson some alcohol, and there appeared to be something white in the bottom of the tumbler. Witness did not know what it was. Mrs. Manson gave Mrs. Wilson a half tumblerful of alcohol. There was some left which the witness tasted. It tasted sweet, and soon after she vomited.

Dr. Herbert Smith, of New Haven, was called; said he had been of the Yale Medical School. He had made a qualitative and a quantitative analysis of a portion of Mrs. Wilson's liver brought him by Coroner Bill, February 13th. The witness described clearly and minutely the various tests employed in his determinations, and exhibited the mirrors obtained from the Marsh test, showing arsenic deposited on the sides of the tubes. The witness found 3.17 milligrams of arsenic in the whole liver. He proceeded, in answer to interrogations of Attorney Torrey, to distinguish the symptoms of acute and chronic arsenical poisoning. His testimony agreed in the main with that of Dr. Wolff on the same point.

Witness said that in his opinion Mrs. Wilson died from the effects of acute arsenical poisoning. Attorney Torrey read to the witness parts of the testimony of Mrs. Fenimore, Dr. Harvey, and Dr. Frank Converse, relative to Mrs. Wilson's symptoms. The witness said they were consistent with symptoms of arsenical poisoning. The witness produced 1 ounce of arsenic and a pin, and showed the jury that a large medicinal dose of arsenic could be taken up on a pin. Witness further stated when asked about Dr. Wolff's opinion as to two fatal doses of arsenic having been administered to Mrs. Wilson a short time before her death, that it was a possible conclusion, but not an absolutely necessary one.

Cross-examined by State's Attorney Hunter, witness said there was no doubt that Mrs. Wilson suffered from chronic arsenical poisoning, as well as acute. On the redirect examination, witness testified that arsenic was practically tasteless, though to some it tasted sweet.

The remainder of the testimony is taken up with the proofs of whether the evidence would show that Mrs. Wilson purposely un-

dertook to commit suicide, and would have no bearing upon toxicology. The jury disagreed upon conviction of the allegation of murder, seven for acquittal and five for murder in the second degree. The jury was then discharged by the court.

September 16th, 1903, Mrs. Manson was again put on trial for the murder of Mrs. Wilson, and without any particularly new evidence except the introduction of an expert witness on handwriting; she was convicted on the charge of manslaughter, and sentenced to state prison for ten years.

So far as the question of toxicology is concerned, there was really no evidence additional to what was brought forward at the first trial.

856.—Case VIII. Trial of Sarah Jane Robinson for the Murder of Prince Arthur Freeman.

(Summary of official report, authorized by Massachusetts Legislature. See footnote 20, p. 31, *ante.*)

This case was for the poisoning of a child of seven years of age by means of arsenic; and the alleged motive of causing his death was for the purpose of securing the value of a life insurance interest on his death. Incidentally, it appeared at this trial that the accused was concerned in causing the death of five other persons, for which crime the grand jury had found indictments, all of which were abandoned except the one above mentioned. This victim was a nephew of the accused; the others were, respectively, her husband, her daughter, her son, her brother-in-law, and her landlord. The case was tried before Justices Field and Knowlton in the supreme court of Massachusetts on the 12th day of December, 1887. Incidentally, the other cases were brought forward in the trial to attempt to show that the motive for the killing of the nephew was deliberate, and for the purpose of her obtaining the insurance money which would pass to her nephew if he lived. There are several interesting points of interest to the toxicologist, which were brought out in the evidence adduced during this trial, and which are of great importance; some of them refer to the complication arising from the use of embalming fluids injected after death into the bodies of the victims, some to the fact that death occurred after a commencing convalescence from an attack of pneumonia, some to the filing of certificates of death for another cause than that of the poisoning, some to the evidence that the vomitus and liquids which appeared upon the clothing of the deceased and upon the bedclothes were not reserved for examination by the experts called to investigate circumstances connected with the deaths of the various victims, some to the question as to whether the arsenical

poison might not have been received by the victims in an accidental way, either from the iron works in which one of them was a workman, or from arsenical wall papers. We invite our readers to examine the details of the published report of this trial.[1]

Embalming fluids.—The first of these victims, her sister, for whose death no bill of indictment was presented, died on the 26th of February, 1885; and before the body was cooled it was given in charge of the undertaker. He found the corpse distended with gas to such an extent that he tapped the abdominal wall to let off the gas, "and the swelling went down;" he "injected considerably less than a pint of embalming fluid." On being asked whether this embalming fluid contained any arsenic, he replied, "I don't know. I don't make, but buy."

The chemical expert, Prof. William B. Hills, an instructor in the Harvard Medical School, testified that he received the organs in four jars. One contained the brain; the second contained the heart and lungs and liver; the third, the stomach, kidneys, and spleen; the fourth, the intestines and contents. He said he found large quantities of arsenic and also mercury. He answered the following hypothetical question: "Assuming Mrs. Freeman had pneumonia; the symptoms had disappeared, were under control, and then new symptoms set in; that she had diarrhea and headache, burning sensation in the throat and stomach, with pain in her stomach, with vomit, and she had died afterwards, and you had found in the organs the amount of arsenic you found in her organs,—should you be of the opinion that she died from arsenical poison? *A.* I should, in the case as you have stated it."

Cross-examination: *Q.* You assume, I suppose, that the arsenic found in the system in that case would have been received into the system during life? *A.* Yes, sir. *Q.* Assuming that it appeared that, upon the death of Mrs. Freeman, a quantity, say a pint, of some preservative or embalming fluid had been injected into the body, and that it was not known whether the fluid contained arsenic or not, would that qualify your opinion? *A.* Yes, sir, it would; because I should not be able to say that such person positively died from arsenic. All that I could say under those circumstances would be this: The symptoms would be consistent with death by arsenic. But I should not draw any conclusion under those circumstances from the chemical analysis. *Q.* Would it be safe to draw any conclusion whatever from

[1] The official report of the trial of Sarah Jane Robinson for the murder of Prince Arthur Freeman, in the supreme judicial court of Massachusetts, published by the attorney general, under chap. 214 of Acts of 1886: Boston: Wright & Potter Printing Co. State Printers, 1888, pp. 112-115.

a chemical analysis, or from the presence of arsenic in the body, if it appeared that the body had been treated with an embalming or preservative fluid which might have contained arsenic? *A.* I think not.

It should be noted, in commenting upon this line of evidence, that the government was dealing with a case of supposed poisoning by arsenic, of a person whose body had been buried for nearly two years; also, that this body had been embalmed before its burial and that it was not in this case necessary to prove the death by poison, because there was another case in which the suspected murderess had administered the same poison, arsenic, to another individual, the proof of which was much more easily made. Had Mrs. Freeman's death been the only suspicious case, it would have been extremely difficult upon the evidence presented to have convicted her.

Arsenical poisoning complicated with an attack of pneumonia.—Here, again, we meet with a difficulty in forming an opinion, from the fact that suspected poisoning by arsenic may be overshadowed by symptoms of a disease, which deceived the medical attendant to such an extent that he certified that the cause of death was from a natural disease, pneumonia, notwithstanding the fact that, upon the evidence submitted at the trial, he was obliged to admit that he had been mistaken, and that, in his opinion, she had probably been the victim of poisoning by arsenic.

Preservation of the vomitus on clothing and bedclothes.—We have particularly mentioned that it is of great importance (see *ante,* § 10) to preserve all articles upon which any vomitus or other organic matters may be deposited, and which may contain particles of poisonous substance, and which are to be submitted to the chemical expert. In this case, as appeared later during the trial, a great opportunity was lost; some of the witnesses in this case testified that the victim vomited on to her clothing, and on the bedclothes, and also on the carpet. None of these articles were preserved, probably because the medical attendant had no suspicion of an attempt to poison his patient. In this connection, it would be well if all medical men who are called to a sudden case where the symptoms are so striking, as in this which we are reviewing, would bear in mind that, possibly, they may be dealing with a suspicious case of poisoning, and, therefore, they should observe certain precautions which later on may be very important; the suspicion can do no harm to their patient. The writers have noticed that in many of the recorded cases which they have read this omission is more common than in their opinion was justifiable under the circumstances.

Could the arsenic which was alleged to have caused the death of the

victim have been derived from some accidental source.—It is interesting to note in this connection that Prof. Hills could not be made to say in his testimony that in an embalmed body the presence of arsenic, when found, was indicative of having been administered before death; while, *per contra,* Prof. Wood testified that its presence under these conditions might be inferred: on account of its unequal distribution throughout the various tissues of the body, that the presence of arsenic recovered from an embalmed body was injected post-mortem; and that, if the amount of arsenic found in the body was uniformly distributed throughout its organs, the inference might be drawn that the poison was received and absorbed before death; in other words, the effect of embalming would be distributed by a slow and unequal diffusion after death when injected into a dead body, while, if administered during life, it would pass into the tissues of the organs by the living processes of absorption.

Prof. Hills gave his opinion that, from recent observations, bodies lying in arsenical soil do not imbibe arsenic in their dead tissues.

The defense attempted to show that the contention of the government, that one of the cases adduced to prove the motive of the accused as a deliberate action to poison her victim, was ill founded, even if arsenic had been found to have been administered before death; because it might have been received and absorbed by the individual from the vapor of arsenic in iron works where the man was employed, or from the vapor emanating from wall papers containing arsenic either as a dye or a mordant. As for the former being the origin of the source of the poison, it was shown by the chemical expert that there was not enough arsenic used in the processes of the iron works to account for the fatal dose; as for the wall paper furnishing the arsenic, Prof. Wood stated in his cross-examination that he had known of two cases of poisoning from arsenical wall paper to which actual poisoning might reasonably be attributed; these two cases had been under the peculiar conditions that there was a large amount of arsenic present, a great many grains to each square yard of the paper, and that, in the apartments on which this wall paper was attached, the air was confined and moist for a long period of time; this would predispose to the evolution of sulphydrate of arsenic. This is a form of arsenical vapor to which accidental arsenic poisoning is attributed. He knew of no other cases of accidental arsenical poisoning, except when the arsenic is heated.

The following important point was developed in the trial of this case: It was shown in the evidence that during the illness of one of the victims there were periods of exacerbation of the symptoms of

poisoning; and during certain intervals prominent symptoms of arsenical poisoning were absent. Prof. Wood stated in his cross-examination that there have been instances of arsenic poisoning in which the symptoms were intermittent, and that these were not uncommon, when the patient would be apparently free from these symptoms for quite an interval; and that afterwards there would be a recurrence of arsenical symptoms without any fresh arsenic being taken. In answer to the question by the counsel, "Owing to the effect of the poison?" he replied, "Probably due to the fact that the inflammatory symptoms begin to subside, and then the secondary symptoms come on a few hours later."

857.—Case IX. Poisoning by Sulphate of Copper.

(Compiled by Assistant Professor Charles Harrington, M. D., of Harvard Medical School.)

(Maschka's Handbuch der gerichtl. Med. Vol. II. p. 291.)

Franz H., sixteen years of age, took, on October 11, 1870, an unknown amount of powdered sulphate of copper mixed to a paste with water. Half an hour afterwards he was carried to the hospital, with severe vomiting and great weakness, thirst, contraction in the throat, choking, and pain in the epigastrium. The latter was sensitive on pressure. The face was pale, the lips blue at the angles of the mouth, the tongue coated and bluish. The temperature of the skin was diminished, the extremities cold, nails cyanotic, pulse small and increased in frequency. The vomitus was blue, and contained particles of the poison. He had several loose, greenish-yellow stools containing no blood. Urine scanty; blood and albumin absent. During the night he was very restless; on the next day complained of very severe headache, pain in the epigastrium, and burning in the mouth and œsophagus. There was no more vomiting. Urine was scanty, and contained albumin, blood, and bile pigment. The patient went home, but was again brought to the hospital on October 15th. He had marked icterus, pallor of the mucous membranes, diminished temperature, frequent pulse, great weakness, cardiac oppression, restlessness; the stools were pappy, brownish-red, and streaked with blood; tenesmus, very bloody urine. The region of the stomach and liver sensitive; liver enlarged; abdomen retracted. The jaundice decreased somewhat up to the 18th. There was then apathy, cold sweat, collapse, and death.

The autopsy was performed two days after. The skin was pale with a tinge of yellow. Rigor mortis in the lower extremities.

There was not much blood in the sinuses, or in the brain and its membranes. The veins of the neck contained but a few drops of fluid blood. The mucous membrane of the trachea and of the œsophagus was pale. The left lung was pale; edematous in the upper lobe; the lower lobe was swollen, hard, and friable, and contained a quantity of foamy fluid. The right lung contained the same kind of fluid. The heart and great vessels contained almost no blood. The heart substance was pale, soft, and easily torn. Stomach and intestines distended; the serous coat of the same, slate-gray in color. The spleen was normal. The liver was of normal size; its substance yellowish-brown in color, soft, easily torn, fatty, and moderately full of blood. The gall bladder contained a few drops of dark, tenacious bile. Kidneys swollen, the cortex yellowish, the pyramids pale brown. The bladder contained some urine; mucous membrane normal. The coats of the stomach were firm, the coronary vessels only moderately injected. The stomach contained a kilogram of brown, feebly acid liquid. The mucous membrane was swollen, thickened, and covered with tenacious mucus; along the greater curvature it was greenish in color. In the fundus was an eschar as large as a cent piece, where the mucous membrane was dirty brown, villous, and easily detached. On detaching it, the muscular coat was exposed. Otherwise there was nothing abnormal about the mucous membrane. The mucous membrane of the small intestine was pale, and not abnormal; that of the large was swollen and pale gray. The large intestine contained some thin, slimy feces.

858.—Case X. Poisoning by Sulphate of Copper—Hemoglobinuria—Death—Autopsy.

(Compiled by Assistant Professor Charles Harrington, M. D., of Harvard Medical School.)

(New York Medical Record, May 27, 1882.)

A well-nourished woman, aged forty-six, took about one ounce of sulphate of copper dissolved in tea, at 9 P. M., December 13th, with suicidal intent. As soon as she had swallowed it, she felt a burning sensation in her stomach and all over her body, and, becoming alarmed, she told a neighbor, who summoned a policeman. He took her at once to a station house. On the way she vomited three or four times, and, before reaching it, began to feel very weak and to suffer from severe pain and cramps in her stomach. At the station house she was given stimulants, and, on the arrival of the ambulance surgeon, large draughts of warm water and flour. This produced free

emesis. At 11 P. M., two hours after taking the poison, she was taken to Bellevue Hospital.

On admission she was very weak and unable to stand or to walk. Her surface was cool and dry; pulse full, strong, 112 per minute; respiration natural, 24 per minute. She complained of faintness, and of cramps in her stomach and legs. She was put to bed at once; and the tube of the stomach pump having been introduced, the stomach was washed out with warm water. It was noticed that the material first obtained from the stomach was grayish-green in color. This was afterwards found to contain copper. After a few minutes, yellow ferrocyanid of potassium was obtained, and 20 grains were added to 1 pint of warm water. This was thrown into the stomach, and, on being drawn out, the water was seen to have assumed a brown color indicating that the chemical change to ferrocyanid of copper had taken place. This injection was therefore continued, the amount of the potash salt being decreased as the brown color became less marked. When the water returned perfectly clear and colorless, the process was stopped. During this time (about forty minutes) the patient had complained constantly of pain in her stomach and of cramps in her legs, and had had several fluid evacuations. Before the tube was withdrawn 3 ounces of a mixture of castor oil and olive oil were thrown into the stomach. A portion of this was rejected. Patient was then given ½ ounce of whisky, hypodermically, as the pulse had become more weak, small, and rapid—128 per minute. She was ordered 20 grains bismuth in milk, 4 ounces every two hours.

December 14th.—Patient vomited several times during the night, and had three loose, brown movements not containing blood. She complained much of abdominal pain and of cramps in her legs. This morning she had less pain, but is very tender over the epigastrium. The cramps occur at longer intervals. She complains of frontal headache and feels very weak. There is no febrile movement, and her pulse is regular, strong, 100 per minute; respiration normal; skin cool and dry. Her mental condition is good; no affection of the senses; the pupils are normal and react to light. Ordered poultices to abdomen; a mixture of bismuth in mucilage; and small amounts of milk frequently repeated. During the day the patient was quite comfortable, did not vomit, had less pain, but had several diarrheal movements attended with tenesmus. Her urine was diminished in quantity, but, as it was passed with the movements, could not be measured. Its color was normal.

December 15th.—Patient had a quiet night and slept. This morning she had some pain and tenderness in the epigastrium, but has not suffered from cramps since yesterday afternoon. She has not vomited, and to-day takes her milk readily. She had, during the night, and continues to have to-day, occasional painful fluid movements, small in amount, brown in color, containing gray masses of feces, but no blood. Her headache continues. At noon, to-day, she complained of pain in the hypogastrium, and began to pass small amounts of urine frequently. Micturition was attended with burning pains in the urethra. The urine was found to be very dark, reddish-black in color, almost like ink. It is turbid, does not transmit light, has a specific gravity 1.014, is acid, and contains a large amount of albumin. On boiling, the coagulum was lighter than ordinary albumin, and floated to the top of the specimen. The substratum of urine was changed to a lighter color. On the addition of acetic acid, the albumin was partly dissolved, and the substratum of liquid regained its original color. Heller's test also demonstrated the presence of albumin. Microscopic examination of the thick sediment, which collected on standing, showed the presence of a large amount of fine, granular matter stained brown. This was in irregular masses, and also in the shape of casts. There were a few epithelial casts, and some epithelial cells from the kidney. Careful examination failed to detect the presence of any blood corpuscles. A portion of the granular matter was dried upon an object glass, common salt was rubbed into it, a hair laid across, a cover glass applied, and a drop of glacial acetic acid allowed to enter beneath the cover. The slide was then warmed, and subjected to examination under the microscope with power of 600 diameters. Crystals of hematin were to be seen, though not in great numbers. A specimen was then examined with the spectroscope, when the two absorption bands between D and E, characteristic of oxyhemoglobin, were distinctly brought out. These tests were sufficient to establish the fact that the patient had developed hemoglobinuria. Microscopic examination of the blood showed a slight relative increase of white corpuscles. The red corpuscles were not decolorized, and no microcytes were visible. The same treatment was continued, with the addition of small doses of opium to allay the abdominal pain and control the action of the bowels.

December 16th.—This morning the patient is found to be moderately jaundiced. She passed a quiet night: had no movements, and did not vomit. She made about 45 ounces of urine during the past

twenty-four hours; appearance and contents the same as yesterday. To-day the abdominal tenderness continues, but is less marked over the hepatic region than elsewhere. There is no fever; but the pulse is more rapid, 120 per minute, and is now quite small. Respiration is also increased to 30 per minute. Her general condition is better than yesterday, on account of the cessation of all gastric symptoms and of the diarrhea. She takes her milk with relish. She is perfectly conscious and rational, but is more quiet, and seems inclined to sleep. The only complaint is that of painful micturition. When questioned, she says that she has slight headache. Opium stopped.

December 17th.—Patient had a restless night, and refused nourishment. This morning she seems to be weaker, and is rather somnolent. She complains of frontal headache, and of pain in the lumbar regions, but says that the abdominal pain has ceased. The abdomen is relaxed, not tympanitic, but pressure develops tenderness all over it. She has not vomited, and has had no movement during the past twenty-four hours. She retains her milk, but has no desire for food, her tongue being still thickly coated and gray. Her throat is not sore. Eyesight and hearing are perfect, and sensation good. Her pulse is feeble, small and rapid, 120 per minute, respiration 30, and, for the first time since admission, there is some fever, temperature being 100.5 °. There is a slight increase in the jaundice, the color of the skin being a peculiar grayish-yellow. She passed 50 ounces of urine during the past twenty-four hours. It presents the same chemical characteristics, and is almost like black ink in color. Her motions indicate weakness, but there is no paralysis.

Toward evening it was evident that the patient was failing rapidly. Her mental condition was stupid, so that she could not be induced to make various motions that were desired to test co-ordination. There was evidently general paresis, as she could no longer turn herself in bed or hold a cup. Sensation was not impaired. At 4 P. M., temperature 101.25°; pulse very small, but regular, 128; respiration, 34. Stimulation was given by the rectum, but was rejected, and the discharge was followed by a movement containing semisolid black feces. She is now unable to swallow. At 9 P. M. the patient had sunk into a semicomatose condition, and, when aroused, seemed unable to make any voluntary motions. Sensation was decidedly impaired, or else she was too stupid to notice irritation. Pupils were contracted, and no longer reacted to light. Nutritive enemata were not retained, and the urine was passed unconsciously. Temper-

ature 98.75°, pulse 140, respiration 40. From this time she lay in a state of coma, and at 2 A. M., December 18th, died.

Autopsy twelve hours after death.—Body.—Rigor mortis extreme. Whole surface uniformly jaundiced. No ecchymoses.

Brain.—The membranes were tinted a grayish-yellow color. A considerable amount of clear serum was present beneath the pia mater. No meningitis. No marked congestion. Surface of the brain was a pale gray color, evidently tinted like the membranes. Brain was wet, but there was no excess of fluid in the ventricles. Substance normal. Cord not examined.

Thorax.—On opening the body it was noticed that the muscles, connective tissue, fat, and all the organs presented a grayish-yellow appearance. All the blood in the body was firmly clotted in the veins. The clots were of a light brownish-red color, resembling chocolate, and very consistent, so that they retained their form on being taken out of the veins. There was no fluid blood in the body.

Pericardium.—No adhesions, no excess of fluid. Heart, normal size. Both ventricles were filled with dark clots. Heart muscle flabby, and, on section, fatty. A mottling was noticed beneath the endocardium, most marked on the papillary muscles. Microscopic examination showed well-marked fatty degeneration of the muscular tissue. Aorta atheromatous.

Pleuræ.—Old adhesions on both sides. No fluid in the cavities.

Lungs.—Slight yellowish-brown coloration of the surface of the lungs. No subserous ecchymoses. Both lungs extremely edematous. The fluid squeezed from them was yellow in color, substance of lungs normal.

Abdomen contained no fluid. Spleen normal in size and consistence. Stomach contained a large quantity of semisolid material, greenish-white in color, apparently half-digested milk. This was tested, and contained no copper. There was but slight evidence of gastritis, consisting only in mild congestion. No ecchymoses and no ulceration.

Œsophagus.—Pseudo-membranous patches of gray color, and ulcerations near the stomach.

Intestines.—Duodenum empty. It was deeply stained with bile. The ductus communis choledochus was pervious. Jejunum in its upper three quarters was free from inflammation. It contained green semisolid masses. Ileum contained thick, hard masses, green in color. These masses did not give any reaction on the addition of

aqua ammonia. For 16 inches along the lower part of the ileum there was extensive ulceration, with the formation of sloughs, and loss of substance on the mucous coat. The ulceration was unevenly distributed through the ileum, and not confined to the region of Peyer's patches. It extended upward into the jejunum, but was less marked than below. The large intestine contained large masses of pasty, dark-green feces, which distended it greatly. When these were washed out, numerous losses of substance were seen on the surface of the mucous membrane. There were no distinct ulcers.

Liver.—Uniform, brownish-yellow color, soft in consistence, normal size, somewhat fatty. Microscopic examination showed extensive fatty degeneration of the hepatic cells. The fat existed in large globules, and not in small molecules. Gall bladder was moderately distended with very thick, black bile; this bile was found to contain copper. The bladder contained a small gall stone: gall duct and bile ducts not obstructed. Pancreas normal. Kidneys slightly enlarged; capsule adherent; surface mottled with very dark brownish-red and gray colors, giving a marbled appearance; substance soft. Cut surface nearly uniform, dark brownish-red, and little distinction to be noticed between the cortex and medulla. Cortex was swollen, and striated appearance not present. The mucous membrane of the pelvis was gray in color, was coated with a thin layer of mucus, and studded with minute ulcers, round in shape. Microscopic examination showed the tubules of the kidneys to be filled with granular matter stained red, and similar to that found in the urine. The cells lining the tubules were swollen; the Malpighian tufts were compressed, and a free space existed between the tuft and its capsule, such as is said to appear when a large amount of albumin has been excreted. The capsule of the tuft was moderately thickened; the capillary walls were thickened; no granular matter in the vessels. Ureters normal. Bladder contained some dark red urine. The mucous membrane was uniformly stained a pink color; no cystitis.

Uterus.—At each cornu, small intramural fibromata. The Fallopian tube on the right side was distended with dark red fluid; its fimbriated extremity obliterated; on the left side there was a small cyst in the broad ligament.

The medico-legal importance of the case warrants its publication. The number of cases of sulphate of copper poisoning reported is small, and in but a few is there a record of an autopsy. In no reported case is there any mention of the occurrence of hemoglobinuria.

859.—Case XI. Fatal Poisoning by Chromate of Lead.

(Compiled by Assistant Professor Charles Harrington, M. D., of Harvard Medical School.)

(Vierteljahrsschrift für gericht. Med. und öffentl. Sanitätswesen, Bd. XXI. p. 60.)

On March 14, 187–, between 9 and 11 o'clock in the forenoon, two young boys, aged respectively one and three quarters and three and a half years, ate a number of small yellow objects, which had been used for decorating a cake formed like a bee hive, and which represented the bodies of bees. In the afternoon, between 2 and 3 o'clock, both were seized with violent vomiting and great prostration. The vomitus was at first yellow in color; it was unfortunately thrown away. The vomiting continued until 11 o'clock, but, during the last three hours, was not so frequent. At 6, in the evening, when the physician arrived, the two were in bed, and presented a very sick appearance. Their faces were much reddened; they complained of much thirst, and were very restless. There was neither diarrhea nor pain. It was not unreasonable to ascribe these symptoms to poisoning, for the children had had nothing but bread and milk for breakfast, and meat soup, beef, and potatoes for dinner, of which the other members of the family had eaten and had remained well. The poison was attributed to the above-mentioned "bees," of which they had been given seven, of which number but one remained. They admitted that they had eaten the other six.

Analysis of the "bees" conducted by an apothecary showed them to be composed of gum tragacanth and chromate of lead.

The treatment consisted, at first, of the administration of calcined magnesia in mixture. On the following day (March 15th), the faces of both were red and hot; they were listless, and, on being questioned, showed signs of discomfort, but made no complaint of local pain; the sensorium was apparently affected. Carl, the younger, had some diarrhea, and towards noon convulsions, during which the face was livid. The convulsions were more frequent towards evening. Ice was applied to the region of the stomach, and given in small amounts internally.

On the 16th, at 9 o'clock in the morning, the younger child died. The elder had constant redness and heat in the face, was listless, and almost stupefied. The skin of the breast and abdomen was markedly erythematous. The evening temperature in the axilla was 39.5° C. Ice and bicarbonate of sodium solution were given internally; ice poultice applied to the gastric region.

On the 17th, the pulse was irregular and intermittent; there was Cheyne-Stokes respiration. Evening temperature 39.6° C. Treat-

ment: Ice as before, and subcutaneous injection of quinin. The latter was given because, in spite of great thirst, deglutition was extremely difficult.

On the 18th, the erythema was still present; bad smell from the mouth; stupor; difficult deglutition. Evening temperature 39.2° C. Treatment as on preceding day.

On the 19th, collapse; very foul odor from the mouth; deglutition almost impossible; sensorium much affected. Death occurred at 11 o'clock in the forenoon, five days after ingesting the poison. Autopsies were performed in both cases. Among the post-mortem appearances observed on examination of the body of the younger child thirty-nine hours after death, the following may be noticed:

The blood vessels on the surface of the brain were strongly injected; the longitudinal sinus empty; blood vessels of the base of the skull overfilled. On section of the brain tissue, single bloody points were seen.

The lungs were congested; on the surface were some small points of emphysema. The heart contained much blood, fluid and coagulated. The intestinal canal was pale, and only occasionally marked by single injected blood vessels. The liver was bright colored, with pale and irregular spots exteriorly and interiorly; on microscopic examination, a marked fat formation was seen,—especially at the above-mentioned spots (fatty degeneration). The stomach contained a small amount of yellowish-green fluid. Its inner surface showed velvet-like opaque swelling of the mucous membrane, especially marked on the cardiac half. The mucous membrane was marked throughout with red points, which, in some places, were grouped; in the vicinity of the cardiac orifice it was pale yellow, and the color could not be wiped off. The duodenal mucous membrane was pale and in folds; occasional bloody points were seen. The spleen was large and of normal color. The kidneys were normal. The mucous membrane of the bladder was pale; the bladder was empty.

The autopsy of the second child was performed twenty-seven hours after death. The skin of the face was yellowish in color. The blood vessels of the surface of the brain were very full; the longitudinal sinus contained but little blood; the blood vessels of the base of the skull were full. The brain substance contained much blood. The dura mater was very adherent.

The lungs were pale red; blood vessels of the heart very full. The œsophagus was much injected and brownish-red; its mucous membrane was disintegrated and purulent throughout, the destruc-

tion being most marked at the upper end. The mucous membrane of the larynx and upper portion of the trachea was purulent and partially disintegrated; the tissues beneath the mucous membrane were deeply reddened. In the right tonsil, which contained much blood, was a pus cavity. The walls of the stomach were somewhat thickened; the mucous membrane at the greater curvature was in overlying folds and colored dirty, pale red; at the pyloric end the color was brownish-red with darker spots. The mucous membrane was loosened and easily rubbed off. On careful examination numerous small blood points could be seen under the mucous membrane; these were grouped at various points, and caused the redness. The mucous membrane was already disintegrated in several places.

The mucous membrane of the duodenum was ulcerated, loose, and easily rubbed off. At the upper part it was strongly injected, and at one place there was a perforation as large as a lentil; at several points the wall was almost perforated.

The liver was fatty degenerated. The spleen was 11 centimeters long; the exterior and the cut surface were light brown; the consistence was soft. Both kidneys were very rich in blood, and, on section, showed small pus drops, which apparently came from the calices. The bladder was very full; its mucous membrane strongly injected.

The chemical analysis of the organs gave in both cases perfectly negative results. Analysis of the bees showed them to consist of 0.270 gram of gum tragacanth and 0.0042 gram of chromate of lead. As has been said before, the number of bees ingested was probably six, and, allowing to each an equal share of the same, the amount of the poison ingested by each was 0.0126 gram.

860.—Case XII. Fatal Poisoning by Inhalation of Dust from Yarn Colored by Chromate of Lead.

(Compiled by Assistant Professor Charles Harrington, M. D., of Harvard Medical School.)

(Vierteljahrsschrift für gericht. Med. und öffentl. Sanitätswesen, Bd. XVII. p. 29.)

A few days before January 1, 1876, a weaver, B. in E., bought a lot of yarn colored by chromate of lead for manufacturing blankets. Some of the yarn was wound on bobbins before New Year; and, on the 2d day of January, B. and his journeyman G. began to work with the same at two looms, while Frau B. and the bobbin girl L. continued the winding. They worked from between 7 and 8 o'clock in the morning until after 10 at night. During the work the yarn gave off

so much dust that, according to B.'s declaration, their faces and hair were quite yellow, and they had a bitter taste in the mouth. Their expectorations were colored yellow. Frau B. called the taste "sweetly bitter." The journeyman became sick about eight days after beginning the work. He was seized with headache, ringing in the ears, pains in the chest and stomach, loss of appetite, inclination to vomit, and constipation. He was ill six weeks, when he was obliged to give up work and consult a physician. Meanwhile, Frau B. was seized with pains in the chest and loss of appetite; but these symptoms disappeared after continued drinking of warm milk.

In the third week after beginning work, B. was taken with pains in the spine, nausea, proneness to vomit, weakness and loss of sleep. Eight or ten days afterwards, he was obliged to cease work and call in a physician, on account of severe cramps in the abdomen and constipation. The bobbin girl, also, was sick with diarrhea, loss of appetite, and pains in the chest.

The symptoms of B. and of his journeyman were the same; yellow coated tongue, yellow sputa, complete loss of appetite, nausea, occasional vomiting, pain in the abdomen,—especially in the umbilical region,—general weakness, and obstinate constipation. The feces were colored yellow. The physician gave as his opinion that these symptoms were caused by inhalation of the dust, and were characteristic of chronic lead poisoning. A weaver, in another town, who had used the same kind of yarn at the same time, suffered from similar symptoms, and became unfit for work. The above cases improved gradually, and the patients recovered; but B.'s nine weeks' old boy was taken sick at the same time, and died in consequence of inhaling and swallowing the dust. This child was, at the beginning of the work, about fourteen days old. On noticing the dust given off from the yarn, the parents sought to protect the child from the same by covering its face with a white woolen cloth, which, however, did not in any way hinder respiration. This cloth and the child's bed were said always to have looked yellowish. The basket in which the child lay stood by day between the two looms, which were about six yards apart. At night the child was taken up stairs into its mother's bed. The child had been active and healthy from birth, and, in spite of the fact that it could not be suckled, had thriven well on suitable food, so that there was no ground for ascribing the post-mortem appearances of the stomach to ill-nourishment, etc. About seven weeks from the commencement of the spinning, the child suddenly became very sick. It had, at first, pallor of the face, and, in a few days, elevated temperature over the whole body. It had several yellow diar-

rheic discharges daily with restlessness and frequent screaming, during which it dug its hands under the pillows. With these screaming fits appeared reddening of the breast and abdomen. At first it would drink, but refused food; later on it drank with some effort, and, on the day of its death, swallowed with difficulty. The lips were dry, respiration quickened, and death came slowly. Neither domestic remedies nor medicine were given the child, partially on account of the popular belief among the laity that not much should be done for small children, but more particularly because the parents had considered the child to be perfectly protected by the cloth from any injurious effects of the dust. Death occurred on February 24, after from six to eight days of illness. It is remarkable that the child, during six weeks' exposure to the dust, remained well, and only six or eight days before death was taken suddenly ill, and grew worse just when no work was going on. The cloth was kept over the child up to the time of death, and it is probable that the cloth became gradually impregnated with dust, which, on sifting through, was inhaled by the child until death. A proof of this is that, on chemical examination after the autopsy, the dust was found in the hair and in the jacket of the child, and, moreover, 0.036 gram of chromate of lead was found in the respiratory tract and œsophagus. The rubber nipple which the child had used was found free from chromate of lead, most probably because the dust which had adhered to the same was sucked off and swallowed. The yarn, on analysis, yielded 11.83 per cent of chromate of lead.

Among the post-mortem appearances the following may be noticed: The hands were clenched convulsively. The omentum was finely injected; between the fundus of the stomach and the spleen was about half a coffeespoonful of chyme; in the fundus a hole about the size of a large pea. The stomach contained chyme similar to that mentioned above. The stomach had a jelly-like character, especially marked about the perforation; the mucous membrane of the right half was much corrugated (rugæ), and had a pale red color, the left half was everywhere softened and smooth, and its color was in some places bluish and in others pale coppery. The blood vessels of the upper curvature were somewhat filled with blood; those of the greater curvature more so. The duodenum contained a light yellow, thin mucus, the jejunum, a pale yellow and watery mucus, and the ileum, a light yellow, thick mucus. The jejunum was pale in color, the ileum marked by very fine vessels, the mesentery of the former was pale, that of the latter injected, and its glands were much developed and hard. The

intestinal mucous membrane showed neither marked reddening nor ulceration. The outer surface of the large intestine was injected; the inner was pale. It contained at the end of the rectum a little yellowish mucus. The liver was normal; the gall bladder contained a teaspoonful of orange-colored bile. Both kidneys—especially the right—contained much blood. The lungs were healthy, and contained much black, fluid blood. The right auricle of the heart, its left half, and the pulmonary arteries, contained black, fluid blood. The brain could not be examined on account of advanced decomposition. The blood in the same was not increased in amount, and was black and fluid.

The chemical analysis of the organs and fluids was negative in its results, excepting in the air passages and œsophagus, where, as has been already mentioned, 0.036 gram of chromate of lead was found.

The death of the child was caused by exhaustion following perforation and softening of the stomach, brought on in consequence of swallowing and inhaling chromate of lead.

861.—Case XIII. Poisoning by Cyanid of Potassium, and Detection of Same in the Body Eight Days after Death.

(Compiled by Assistant Professor Charles Harrington, M. D., of Harvard Medical School.)

(Vierteljahrsschrift für gericht. Med. und öffentl. Sanitätswesen, Bd. XXIX. p. 49.)

On April 19th, in the evening, Von J. entertained a stranger at his inn; the guest not making any appearance on the following day, and the door of his room being locked, the police were summoned. They found the lodger lying dead in bed, and the physician who was called could not state whether he had been poisoned or had died from apoplexy. In front of the bed was found a small bottle apparently containing oil, which was preserved by the police.

The autopsy, which was performed nearly five days after death (April 24), gave the following results: The corpse, which was that of a man of about thirty years of age, strong, and well-nourished, showed no other trace of decomposition than a slight green discoloration of the abdomen. The color of the anterior surface of the body was somewhat yellowish, while that of the posterior surface was light red with bluish-red marbling. Post-mortem rigidity was not much marked in the limbs. The face was somewhat livid, and the eyelids closed; pupils dilated. The mouth was half open, and its mucous membrane was of a pale red color. The nails of the strongly contracted fingers were colored dark blue. On account of suspicions of poisoning, the cavity of the abdomen was the first to be opened,

whereupon, a distinct and unmistakable odor of prussic acid was perceived. There was no abnormal fluid in the peritoneum.

The omentum was dry, and traversed by distended venous blood vessels. The stomach was much distended by gas; and its anterior surface and that of the intestinal coils, which were pale reddish and pale brownish in color, were traversed by a network of numerous, fine, highly injected blood vessels. On the posterior upper surface of the stomach, near the pylorus, was a very even, dark-colored expansion, about as broad as the hand. The contents of the stomach consisted of about 100 grams of a watery, turbid, bloody fluid. The mucous membrane was regularly brownish-red in color and very brawny, but could not be stripped off. It was swollen by imbibition of watery blood, and particularly so at the above-mentioned expansion, where the color was deeper. Erosions were nowhere present; the blood vessels were moderately filled. The stomach and its contents gave off a strong odor of prussic acid, together with a decomposition smell. The small intestine, which, exteriorly and internally, resembled in color, etc., the stomach, contained a small amount of a red, slimy fluid. The vessels of the mesentery and the inferior vena cava were overfilled with dark, almost black, blood resembling cherry juice. The spleen was, outwardly, deep steel-blue; the cut surface dark red. It contained an enormous amount of blood, while its tissues were nearly normal in character. From the cut surfaces of the bluish-red-colored kidneys and brownish-red liver, a thin, almost black, blood flowed. The cut surfaces of the liver and spleen gave off a plainly perceptible odor of prussic acid.

The pericardium was exteriorly traversed by a network of fine, overfilled blood vessels, while the inner surface showed no remarkable injection. The pericardium contained about 20 grams of a yellow, serous fluid. The auricles and ventricles, the coronary vessels, the pulmonary arteries, the venous trunks of the thorax, and the veins of the neck, were full of dark, fluid blood. The tissue of the heart was normal. After removal of the heart, about 300 grams of blood collected in the thoracic cavity. The larynx contained some red, foamy fluid, and its mucous membrane and that of the trachea were of a brownish color and somewhat injected. The much-expanded lungs were dark steel-blue in color; the lower and posterior portions were almost black. They felt elastic, and, on pressure, yielded a sound of crepitation. On the surface were found single bright-colored blood extravasations, circular and elongated, varying in size from a pea to a lentil. The lung tissue throughout contained air,

and from the cut surfaces flowed dark blood and bloody foam, on gentle pressure; a considerable amount flowed without pressure. The bronchi contained considerable, red, foamy liquid; their mucous membranes were moderately injected. The pleural sacs contained no fluid.

The dura mater was bluish, moderately moist, and somewhat glossy; many of the ramifications of the blood vessels on the outer and inner surfaces were quite full. The superior longitudinal sinus contained about 5 grams of somewhat lighter blood. The pia mater was normal, excepting a strong injection of the ramifying blood vessels. The cerebrum was of normal consistence; on its pale red cut surfaces were seen innumerable bloody points and streaks. The brown-red-colored venous plexuses of the empty ventricles were filled with blood. The cerebellum and other parts of the brain were also rich in blood; the blood vessels at the base of the skull contained much fluid, almost black, blood. Directly after opening the skull the odor of prussic acid was unmistakable, and much more so on examination of the brain.

The bottle, which was mentioned near the beginning of this case, contained a few drops of watery, whitish, cloudy liquid, which smelled of ammonia and prussic acid. The presence of ammonia was shown by the white fumes produced on bringing a glass rod dipped in hydrochloric acid to the mouth of the vial; that of prussic acid was shown by the iron test. The further analysis proved the liquid to be a very concentrated solution of cyanid of potassium.

It was decided that the deceased had come to his death by probable poisoning by cyanid of potassium. In order to make this perfectly sure, it was determined to submit the organs to chemical analysis, and, on account of the ease with which the poison decomposes, the analysis could not long be delayed. On the 27th of April, at least eight, and perhaps nine, days after death, the analysis was begun. The stomach and intestines showed, by the color and smell, only moderate decomposition, but the odor of prussic acid was no longer perceptible. The distillate from the contents of the stomach, and portions of the stomach itself, gave the characteristic reactions of prussic acid. The analysis of the liver, spleen, kidneys, and blood, yielded the same reactions. How much of the poison the deceased had taken could not be determined, and the amount found in the body was not quantitated. The microscopical and the spectroscopical examination of the blood gave no important results.

The comparatively slight degree of decomposition five days after

death, the marked rigor mortis, which by a number of authors is stated to be characteristic, the dark color and fluidity of the blood, with the universal congestion of all the organs,—especially the heart and lungs,—the injection of the mucous membranes of the larynx, trachea, and bronchi, and the foamy contents of the same,—are peculiarly interesting, in that, without the appearances of the stomach and the chemical results, the supposition of death from suffocation might have been entertained. It must, however, be further remarked that the dark blue coloration of the lungs, spleen, and kidneys, and also the fine network of injection of the pericardium, have, as far as is known, not been met with in cases of suffocation. The most interesting point in the case is that the poison could be so easily detected at so long a period after death.

862.—Case XIV. Prussic Acid Poisoning from Ferrocyanid of Potassium and Aqua Regia.

(Compiled by Assistant Professor Charles Harrington, M. D., of Harvard Medical School.)

(Vierteljahrsschrift für gericht. Med. und öffentl. Sanitätswesen, Bd. XXVI. p. 57.)

On Sunday April 9, 1876, Udo B. of U., an unmarried merchant, was found dead in his bed at 5 o'clock in the afternoon. On the table was a small vial containing a yellowish liquid, and labeled hydrochloric acid. The deceased was clothed in his shirt, drawers, and socks, and was covered with the blanket. Since he was often heard to express his intention to kill himself, suicide was thought probable. A medico-legal examination was ordered, and on April 11th, forty hours after death, it was made.

On the bosom of the shirt and on the collar were numerous wine-colored and dark violet spots. Rigor mortis was present,—particularly in the extremities and abdominal muscles. The facial expression was calm, the face pale. The skin at the right nostril was brownish-red, rough, and leathery. Beneath the lower lip to the right was a brownish-red spot 1.5 centimeters long, where the skin was dry and leathery. The lips were brownish-red and dry. The inner surface of the lower lip was colored deep red on its right half. The epidermis at these places came away in shreds, on being handled. The abdomen in the gastric region was retracted; lower down it was distended.

The organs of the abdomen were normal as regards position. The stomach was flaccid, and dirty gray in color; on attempting to remove the organ, a great hole was torn near the greater curvature; it contained about 60 grams of dirty brown liquid. The inner surface

of the stomach was colored dirty blackish-gray; towards the fundus uniformly red. The finer and coarser blood vessels were very full; the blood in the veins was coagulated and of dry and smeary quality and black color. The finer veins appeared dotted, on account of the breaking up of the coagula. The mucous membrane was soft and easily torn. The exterior of the duodenum was, for a distance of 12 centimeters from its commencement, uniformly red in color. The duodenal mucous membrane was softened; it was reddened in blotches; its blood vessels were full. In the posterior wall of the duodenum near its commencement there was a place about a square centimeter in area and colored dark-red. In the centre of this spot there was a perforation as large as a pea.

The liver was enlarged, flabby, and pale red; its cut surface was uniformly reddish-yellow; it contained much blood. The kidneys were of normal size and flabby; the capsules somewhat opaque and easily detached; the surface after removal of the capsule was smooth. On section, the cortex and the medullary substance were found to be dark red and filled with dark blood. The lungs contained much blood. The ventricles contained fluid blood of a dark color; the left auricle, a clot dark in color, dry, friable, and circular. The posterior wall of the left auricle was of a slate color, dry and hard.

The tongue was pale; on the dorsum, dirty, brownish-red, dry, and rough. The palate, pharynx, and uvula were white; the mucous membrane of the œsophagus dirty gray in color, soft, easily torn, and strongly injected; the blood in the injected vessels black, dry, and friable. The mucous membrane of the larynx and trachea much reddened; that of the arytenoid cartilages and vocal cords easily detached.

The diploe contained much blood, the longitudinal sinus likewise, the blood dark and fluid. The arteries of the brain were empty.

The brown, leathery spots on the nose and upper lip, the corrosion in the cavity of the mouth, throat, larynx, œsophagus, and stomach, the softening of the stomach, the coagulation in the blood vessels of the œsophagus and stomach, the formation of a peculiar dry clot in the left auricle, and the character of the wall of the auricle next to the œsophagus,—are unmistakable evidences that a mineral acid had been taken.

It transpired that the deceased had vainly endeavored to procure some cyanid of potassium, that he had read works on chemistry, and that he had inquired about the technical use of ferrocyanid of potassium. He had been seen at his window an hour before the discovery

of the corpse, which lay covered in a quiet position. These facts taken together made it strongly probable that death was not caused by mineral acid alone, but by a quickly acting poison.

The chemical examination of the liquid in the bottle showed it to consist of equal parts of hydrochloric and nitric acids. The amount contained in the bottle was 63 grams, and the capacity of the bottle was 100 grams. The contents of the stomach, on standing, deposited a brownish-red heavy sediment. On testing the fluid for hydrochloric acid with nitrate of silver, a heavy, cheesy precipitate was obtained; on performing the test for nitric acid with sulphuric acid and ferrous sulphate, instead of a brown color, a blue precipitate was obtained, which was soluble in hydrate of potassium. A few drops of the fluid were then dried and ignited; the residue was alkaline and partially soluble; the soluble portion contained potassium and sodium, the insoluble was oxid of iron. Some of the fluid diluted with alcohol was distilled, and the distillate was received in a weak solution of nitrate of silver; the resulting precipitate contained cyanid of silver. When almost completely distilled, the smell of prussic acid was perceived. The residue in the retort contained cyanid of iron. The production of Prussian blue on addition of iron salts to the fluid from the stomach, the potassium and iron in the residue after ignition, the forming of hydrocyanic acid on distillation, and of cyanid of iron in the residue, showed that either decomposition products of ferrocyanid of potassium were present, or the salt itself, or both. In order to determine this point the fluid was tested for free prussic acid. For this purpose a part of the fluid was mixed with bicarbonate of potassium, and distilled in an atmosphere of carbon dioxid. Free prussic acid was discovered in the distillate, and the ferrocyanid of potassium in the residue.

The presence of the salt and its decomposition products, and of the acids, shows that the salt and acids had both been employed. Contact of these had formed prussic acid. The deceased had undoubtedly taken the salt in solution; the swallowing of the acids was associated with development of the gas and coughing, whence the eschars on the face and the spots on the shirt and collar. Death must have resulted quickly.

863.—Case XV. Poisoning by Oil of Bitter Almonds (Prussic Acid).

(Compiled by Assistant Professor Charles Harrington, M. D., of Harvard Medical School.)

(Maschka's Handbuch der gerichtl. Med. Vol. II. p. 325.)

A healthy, strong man of twenty-four years drank from a bottle

containing 1½ pounds of oil of bitter almonds, and died in about five minutes. The autopsy was performed in twenty-four hours. Cadaverous smell was hardly perceptible. Rigidity was marked in the extremities. The pupils were moderately dilated. On both breasts and upper arm, in the left groin, and over the whole posterior surface were diffuse, bluish-red blotches (suggillations). The eyelids and lips were closed; the jaws were firmly shut. The sinuses and vessels of the dura mater were filled with black fluid blood. The arachnoid was somewhat opaque. Vessels of the pia mater strongly injected. The cerebrum was rich in blood; the ventricles contained about 15 grams of serum. The plexus was not immoderately full. The cerebellum and medulla oblongata contained considerable blood. The whole brain was hard. The lungs were elastic and normal; they contained considerable dark cherry-red, fluid, foamy blood. The heart was normal; the right ventricle contained about 15 grams of dark fluid blood; the left was somewhat enlarged and nearly empty. The great vessels contained but a small amount of blood. There were no serous exudations in the pericardium or pleural cavities. The stomach and intestines were exteriorly bluish-red; otherwise normal. The kidneys were normal and much engorged. The liver and spleen were moderately rich in blood. The large blood vessels of the abdomen moderately filled. There was no exudation into the peritoneal cavity. From all the large cavities of the body, and from the ventricles of the brain, there arose such an intolerable, sweetish smell that several persons present at the examination vomited, and all present suffered from headache and weakness, which persisted on the following day.

The chemical examination was performed thirty-six hours afterwards, and 0.480 gram of anhydrous prussic acid obtained.

864.—Case XVI. Poisoning by Nitrobenzol. Death in Nine and a Half Hours.

(Compiled by Assistant Professor Charles Harrington, M. D., of Harvard Medical School.)

(Maschka's Handbuch der gerichtl. Med. Vol. II. p. 335.)

Three young workmen prepared a bottle of "Schnaps," which was 1 part alcohol, 2 parts water, and (according to their story) but 20 drops of nitrobenzol. The latter was added to impart a taste of almonds to the mixture. M. G., nineteen years old, drank about a third of the whole amount between 7 and 8 o'clock in the morning. Each of the others took four or five swallows, and three workgirls each took a taste. Of these persons, but one suffered violent abdom-

inal pain; the others remained perfectly well. Shortly after 8 o'clock a blueness was noticed in M. G.'s face; at 9:30 he vomited, and had pain in the abdomen. At 10 he lost consciousness, fell down, and had twitching of the arms and hands. At 11:30 he regained consciousness, took some milk and an emetic, vomited repeatedly, and was carried to the hospital. The skin was cold; pulse 100, weak, and irregular; respiration slow and superficial; complete loss of consciousness; trismus, stiffness of the neck, fibrillary twitchings in the masseters, spasmodic flexion of the arms. The skin was grayish-blue (too gray for ordinary cyanosis), pupils dilated and almost incapable of reacting, lids closed. The eyes rolled very slowly from out inwards. A strong smell resembling that of bitter almonds came from the mouth. Involuntary dejections. The stomach was washed out, by means of the stomach pump, with about 5 quarts of water. The water on its return was milky, and without a distinct smell. He improved somewhat after being bled at 2 P. M. (60 gr.); answered now and then, and could swallow. The blood which flowed from the vein was dark and thin. Under the microscope the blood corpuscles were rather brighter. After 3 P. M. somnolence; respiration irregular. Death occurred at 5:30 P. M.

The autopsy was performed forty hours after death. Rigor mortis marked; numerous red blotches (suggillations) over the whole body. The dependent parts bluish-red; face dirty grayish-yellow. The vessels of the skull were very full; the cortical substance of the brain was comparatively anemic, the medullary substance hyperemic; the pons and medulla were pale. The blood in the jugular veins was fluid and brownish-red. Marked catarrh of the whole throat; the mucous membrane was brownish-red; there was a bluish-red injection of the mucous membrane of the larynx and trachea. Mucous membrane of the epiglottis much swollen. The lungs were full of brownish and black blood. Pericardium empty; right auricle much dilated and contained soft, dark, brownish-red coagula; left ventricle contained but little blood; heart substance dry, hard, and somewhat streaked with yellow. Liver was markedly reddened and contained much dark fluid blood. On incision, a distinct bitter almond smell was perceptible. The spleen was firm and congested. The kidneys showed marked passive hyperemia; considerable cloudy urine exuded from the pyramids on pressure. The stomach was moderately distended; its mucous membrane was covered with tenacious grayish-yellow mucus, which had the same smell as the liver. The mucous membrane was very thick; some imbibition of blood, but no hemor-

rhages. The pancreas was much congested. The serous coat of the intestine had imbibed some blood; the mucous membrane was diffusely reddened and swollen; the single follicles swollen. The bladder was full of clear urine, which had no particular odor.

865.—Case XVII. Poisoning by Aconitin.

(Compiled by Assistant Professor Charles Harrington, M. D., of Harvard Medical School.)

(Lamson Case in England.)

(The Lancet, March 18, 1882, p. 455.)

On Wednesday, March 18th, 1882, at the central criminal court, before Mr. Justice Hawkins, George Henry Lamson, aged twenty-nine, surgeon, was indicted for the wilful murder, on December 3d, 1881, of his brother-in-law, Percy Malcolm John, aged nineteen. Percy Malcolm John, who was a cripple with curvature of the spine and paraplegia, had property to the extent of £3,000, half of which, at his death, would revert to the prisoner's wife. John had, for three years prior to his death, been at Blenheim House School, at Wimbledon, kept by Mr. W. H. Bedbrook. On December 3d, 1881, John was, with the exception of his paralysis, in good general health, and, on that day, had taken his meals—breakfast, dinner, and tea—in company with Mr. Bedbrook among others. On Saturday, December 3d, Lamson called on John at Blenheim House at 6:55 P. M., and their interview took place in the dining room of Blenheim House in the presence of Mr. Bedbrook. Mr. Bedbrook offered Lamson some wine, which he accepted; and Lamson then asked for some sugar, as the wine (sherry) was rather strong, and he said "sugar would destroy the alcoholic effects." A basin of white sugar was brought, and Lamson put some of it into his sherry. Lamson then produced a Dundee cake and some sweets, of which all three partook. At 7:15 P. M. Lamson produced a box of gelatin capsules from his pocket, and said, "Oh, by the way, Mr. Bedbrook, when I was in America I thought of you and your boys. I thought what excellent things these capsules would be for your boys to take nauseous medicines in." Lamson then gave a capsule to Mr. Bedbrook, and, filling another with sugar, handed it to John and said, "Here, Percy, you are a swell pill taker; take this, and show Mr. Bedbrook how easily it may be swallowed." John swallowed the capsule; the prisoner soon said, "I must be going," and at 7:21 P. M. left the house. A little after 8 P. M., John complained of heartburn, and soon after

said, "I feel as I felt after my brother-in-law had given me a quinin powder at Shanklin." He was carried up to his bedroom, and, about 9 P. M., was found in great pain and vomiting. He complained that "his throat appeared to be closing, and the skin of his face felt drawn up." At 11:30 P. M. he died. John was treated by linseed poultice to the abdomen, white of egg beaten up with water, and two hypodermic injections of morphin of 1/6 and ¼ grain respectively.

The post-mortem examination of John's body was made on December 6, 1881, by Dr. Little and Mr. Berry of Wimbledon, and Mr. Bond of the Westminster Hospital. The spinal disease was found to be old and inactive. There were some old adhesions of the lung. The lips and tongue were pale. The cerebral meninges, liver, kidneys, spleen, and the mucous membrane of the stomach, were much congested. The mouth and lips were pale. On the under surface of the large end of the stomach were six or eight yellowish-gray patches, a little raised, about the size of a small bean, and towards the smaller end were two or three similar ones. The heart was almost empty, but healthy. The lungs were congested, the posterior parts very much so.

The analysis of the viscera, vomit, and the articles of which John might have partaken, was conducted by Dr. Stevenson of Guy's Hospital, in conjunction with Dr. Duforé of the Westminster Hospital, and the results obtained by the one were verified by the other. The results were briefly as follows:—

1. Portions of the liver, spleen, and kidneys, treated by Stas's process, gave evidence of slight traces of morphin, and the alkaloidal extract, when placed upon the tongue, produced the numb, tingling sensation which is characteristic of aconite, and which, for the sake of brevity, we will call "aconitism."

2. The contents of the stomach similarly treated produced aconitism.

3. The stomach itself similarly treated showed the presence of an alkaloid, but the extract failed to produce aconitism.

4. The urine gave evidence of morphin and aconitin, and the extract obtained from 1 ounce of the urine killed a mouse in thirty minutes, when injected under its skin; the symptoms being exactly similar to those produced by injecting a minute quantity of a solution of Morson's aconitin.

5. A mixture of the extract from 1, 2, and 3, when injected under the skin of a mouse, killed it with similar symptoms in twenty-two minutes.

6. The vomit was found to contain muscle, starch, onion, vegetable

pulp (probably apple), raisins, candied peel, and pineapple essence. It contained neither morphin nor quinin; but very marked aconitism, which lasted for over six hours, was produced by a minute quantity of the extract, and, when injected under the skin of a mouse, it produced powerful symptoms in two and a half minutes, and killed it in a quarter of an hour.

Dr. Stevenson considered that the vomit contained as much as a quarter of a grain of aconitin.

In the beginning of 1881, Mr. Bedbrook received a letter from Lamson, who was then in America, and a box containing a dozen pills. The letter requested Bedbrook to give the pills to John, as Lamson had heard of cases in America similar to that of John being benefited by the pills in question. The deceased had taken one of the pills, and the next morning complained of being very unwell, and said he should take no more of the pills. On August 28th, Lamson bought 3 grains of sulphate of atropin and one grain of aconitin of Mr. Albert Smith, a druggist of Ventnor. On August 29th, Percy John, who was staying in the house of Mrs. Jolliffe at Ventnor, was taken ill with diarrhea and prostration, and a feeling "as if he were paralyzed all over."

Lamson was living with his father in Ventnor between August 6th and October 23d, 1881, and was in the habit of going to the house where Percy John was living, and had actually called upon him on the 29th. On October 13th, he bought twelve quinin powders (containing a grain and a half each) of Mr. Littlefield, a druggist of Ventnor. John, at the time of his death, was taking quinin powders, which had been supplied to him by Lamson. On November 11th, Lamson had bought of Bell & Co. ½ ounce of a mixed solution of morphin and atropin, containing 40 grains of morphin and 1 grain of atropin and, on the 16th of November, he bought the same amount of a similar solution. On November 16th, he also asked for 5 grains of digitalin, which were not given to him, because the sample in stock was not thought to be good, and on the 20th of November, he asked for 1 grain of aconitin, with which the assistant in the shop refused to serve him. On November 24th, he purchased 2 grains of aconitin of Messrs. Allen and Hanbury.

Among the effects of the deceased were found twenty quinin powders numbered from 1 to 20. Numbers 1, 2, 3, 4, 5, and 6 were in larger papers than the rest, and the powders were nearly uniform in weight, containing about a grain and a half of quinin each. Nos. 7 to 20, inclusive, were wrapped in smaller papers, and varied in

weight from .6 of a grain to 1.8 grains. Nos. 16, 17, and 19 differed in appearance from the rest, having an admixture of a pale fawn-colored substance. They all contained aconitin. No. 16 contained 0.83 grain of aconitin and 0.96 grain of quinin. One fiftieth of a grain of the aconitin contained in this powder killed a mouse in six minutes and a half. One of the pills which had been sent by Lamson from America was found to contain nearly half a grain of aconitin. Some of it injected into the back of a mouse killed the animal in less than five minutes, and the aconitism produced by a small quantity on the tongues and throats of the experimenters lasted for over seven hours.

Witnesses were called to prove that Lamson would benefit pecuniarly by the death of deceased, and that, at the time of John's death, he was a bankrupt without a penny in the world. No witnesses were called for the defense. The counsel for the defense was unable to bring forward any solid arguments in refutation of the evidence and the opinions of the experts, and the jury, after deliberating thirty-five minutes, returned a verdict of guilty. Sentence of death was passed in the usual form.

866.—Case XVIII. Recovery after a Dangerous Dose of Aconite and Chloroform.

(Taken from text in former edition.)

(New York Medical Journal, April, 1875, p. 371.)

The following case of poisoning by aconite and chloroform simultaneously swallowed presents so striking a description of the poisonous effects of aconite that it is reproduced in detail from the report of Dr. John E. Blake: "A young lady . . . took by mistake something more than one dram of a mixture containing equal parts of the tincture of aconite root and chloroform. [The chloroform would probably have no controlling effect upon the deadly action of the aconite.] . . . The burning taste of the chloroform caused the error to be at once perceived. The patient took a potion of mustard and water, and immediately walked to my house, accompanied by a young lady friend, . . . about one eighth of a mile. I allow for walking this distance not more than five minutes, and believe that not more than fifteen to twenty elapsed from the time the dose was taken before I saw her. She had the presence of mind to bring the bottle with her, by which I was at once informed of the

exact nature of the poison, presuming from the word 'liniment' [on the label] that the aconite was of the root, and was probably in large amount. This presence of mind on the part of the patient, and the perfect calmness which never deserted her, and which was also exhibited in a striking manner by her friend, helped me much in this sudden emergency, and contributed to save her life.

"There were absolutely no symptoms at this time. I had in my pocket between 15 and 20 grains of sulphate of zinc, which I gave her. . . . I went down stairs for a stomach pump. Returning to the chamber, and finding that no vomiting had taken place, I introduced the tube of the stomach pump at once, and pumped in something over a pint of warm water. On drawing this water back, it returned so impregnated with the poison that the strong odor of chloroform was very perceptible. The aconite gave the water a dark tinge. I thus passed rapidly through the stomach nearly two quarts of water, and am convinced that none of the poison remained. Enough, however, had been absorbed to do deadly work, and numbness of the tongue and cheeks began to be perceived before the operation of evacuating the stomach was finished, rapidly extending itself over the shoulders and back and down the arms. . . . Stimulants were administered, which Miss C. was able to swallow. . . . I had scarcely left the bedside . . . when the patient, articulating with great difficulty said: 'All is dark now, Doctor, I cannot see at all;' and at once became unconscious. Not more than two minutes before this she had been able, with slight assistance, to totter, with faltering and uncertain step, from the sofa to the bed. . . . Dr. Thomas arrived . . . an hour after the poisonous dose was taken; . . . the patient's situation . . . was, briefly, as follows: Her breathing had become imperceptible, she was growing cold, and she was pulseless. No pulse whatever could be detected, even in the axilla, and she remained without any trace of pulse for a period of over three hours. This is a point about which there can be no mistake, one to which our attention was carefully and constantly directed." By artificial respiration with oxygen gas, by electrical stimulation, and by keeping the head low, the patient gradually recovered from this death-like syncope, the pulse at the wrist showing an almost imperceptible thrill more than three hours from the time the dose was taken. This reaction was succeeded by a comatose condition, with an overexcitability of the skin, a feeble current of the electric battery caused her to cry out. Five hours after the commencement of the poisonous symptoms, some urine was obtained and

examined; this resulted in finding it loaded with albumin and fragments of renal casts. The electrical stimulation was continued at times during the succeeding eleven hours, "since, if discontinued for more than ten minutes, the pulse would flag at once, and coma come on." Some urine, collected again seventeen hours after the beginning of the poisoning, showed copious amounts of albumin and casts, but during the next few days these gradually disappeared. "For several days she could recall nothing whatever of what had passed, and could not imagine how she came to be where and as she was. Slowly, however, all has come back, and she remembers pretty well all that occurred up to the time of her being insensible." She fully recovered. Other cases of poisoning are referred to in the footnote.[2]

867.—Case XIX. Poisoning by Savin.

(Compiled by Assistant Professor Charles Harrington, M. D., of Harvard Medical School.)

(The Lancet, 1845, I. p. 677.)

A young woman, M. A. N., aged twenty-one, had enjoyed remarkably good health up to twelve hours before her death. On the night of Tuesday, April 22d, she supped with her lover, by whom she was far advanced in the family way. She was then cheerful and hearty, but about 3 o'clock in the morning she awoke her mother, with whom she slept, and complained of a violent pain in her stomach. She was very sick, and her mother, believing that it arose from a too hearty meal, got up and gave her some brandy and water. The sickness, however, was not diminished, and she subsequently became insensible. About 8 o'clock her mother sent for the parish surgeon, whose assistant came to see her; he prescribed some ordinary medicines, which, from her insensibility, they were unable to administer. Fearing that there might be some danger, the mother sent for another surgeon, Mr. Newth, who got to her about 11 o'clock. She was lying on her back, perfectly insensible, breathing laboriously, and with stertor; she was foaming at the mouth, her countenance was turgid; the

[2] Stewart, Case of Poisoning with Aconite and Fusel Oil, Edin. Med. Jour. 1879, 904; O'Brien, Case of Poisoning by Aconite, Med. Rec. N. Y. 1879, 128; Carleton, Case of Aconite Poisoning, Bos. Med. and Surg. Jour. 1879, 544; Elliot, Case of Aconite Poisoning, Lancet, London, 1878, II. 917; Pike, *Ibid.* 1880, I. 12; Hardman, *Ibid.* 1882, I. 37; Samson, A Case of Poisoning by Aconite, Chicago Med. Rev. 1882, 91; Van Peski, Empoisonnement par le nitrate d'aconitine, Gaz. Hebd. de Sci. Méd. Montpellier, 1880; Unique Case of Poisoning by Aconite, Brit. Med. Jour. 1881, 64.

eyes shut, with the pupils much contracted; her limbs were also convulsed, and the mother told him that she thought she had been suddenly taken in labor. He therefore made an examination *per vaginam,* but found that the os uteri was only slightly dilated; the membranes, however, were ruptured, and the uterus was still acting vigorously, each contraction being accompanied by a convulsive tremor. Believing it to be a case of puerperal convulsions, he bled her, and applied cold to the head, but this did not in any degree restore her. As the os uteri became more fully dilated, he found that the head of the child presented, and that it was gradually descending. About 3 o'clock, while the labor was thus progressing, she uttered a low moan, and in a few minutes ceased to breathe. Mr. Newth immediately sent for the forceps, and delivered her of a male child, which was dead; as far as he could judge from its appearance and the mother's report, deceased was somewhere between the term of the seventh and eighth month of pregnancy.

Twenty-four hours after death, Mr. Newth made an examination of her body. It bore no marks of violence, was well formed, and rather robust. On opening the head, he found that the vessels upon the surface of the brain were much gorged with black fluid blood, and, in proceeding with sections of the cerebral substance, discovered that the gray matter was infiltrated in various parts by the same dark-looking blood; this he especially noticed in the anterior part of the corpus striatum. The choroid plexus was also gorged; the ventricles were empty. The lungs were a little congested on the dependent parts, but otherwise healthy. He did not examine the heart, nor did he pay attention to any of the abdominal viscera besides the stomach. This, on being opened, appeared rather paler than usual, excepting one or two spots, which were red, as if blood had been effused into the mucous tissue. It contained about four ounces of fluid, which he secured, and took to H. Letheby, Esq., M. B., for examination. This fluid had a dark brownish-green color, smelled of digesting matter, and exhibited an acid reaction. Oil of savin was obtained from the fluid by distillation. A little of the sediment of the original bottle was afterwards examined under the microscope, and compared with the powder of the dried plant; their structure appeared to be perfectly identical. The rest of the fluid was filtered, and the residue digested with ether, by which means there was obtained a green solution, containing resin and the green coloring matter chlorophyl.

868.—Case XX. A Case of Digitalis Poisoning.

(Compiled by Assistant Professor Charles Harrington, M. D., of Harvard Medical School.)

(Vierteljahrsschrift für gericht. Med. und öffentl. Sanitätswesen, Bd. XXIV. p. 278.)

Two young German conscripts, who had, on examination, been declared healthy and fit for military duty, went to a "liberator" to get the means of avoiding the same. He explained to them that healthy recruits, who could not be exempt, but who wanted to be, must be sick; and he gave them a medicine which would bring on a diseased condition—an abdominal affection—in consequence of which exemption would be gained without any permanent injury to health. Each obtained a box containing one hundred pills, with directions to take four pills twice daily. A short time before entrance to the army, perhaps eight or ten days, they began to make use of the pills. They entered the army on December 12th, 1874, and three days afterwards one of the two, G. von K., announced himself as sick, and was treated a few days in barracks, then, on the 18th, admitted to the hospital. After three weeks' treatment, he very suddenly and unexpectedly died. Shortly before his death the officer, under whose command the two recruits belonged, reported that a rumor had spread that the two were employing some means to appear sick in order to avoid duty. In consequence of this rumor, and on account of the inexplicable death, a legal examination of the body was made. The autopsy revealed no sufficient cause of death, and the suspicion of poisoning was strengthened. The finding of a box containing thirteen pills in the stocking of the dead recruit strengthened the suspicion, and the other recruit C. K. was arrested, since he was suspected of using the same means to attain the same end as G. von K. had in view. The recruit C. K., directly after entrance into the army, was declared by the staff surgeon to be temporarily unfit for duty, on account of deficient circumference of the chest, and this report had been confirmed by the commission. In addition to the above defect the sick appearance of the soldier was particularly noticeable. The recruit himself had no certain knowledge as to the real reason of his exemption, and had continued the use of the pills; yet, on account of the appearance of serious symptoms, he had for a day discontinued their use. On receipt of the news that his friend was dead, he threw away his pills out of fright. This he admitted at the examination, when, after a firm denial, he was pressed to an open confession. He admitted that he had used the pills, and described the effects to be exactly like the symptoms of the deceased.

The autopsy was performed twenty-two hours after death, and was negative in its results; the organs and tissues were in a state of health. There were no remarkable external appearances. The body was well nourished; the panniculus adiposus was normal; the muscles were pale. The blood was thinly fluid, nowhere coagulated, and dark cherry-red in color. The organs of the chest were perfectly normal; the lungs and heart floated when placed together in water. The lungs were nowhere adherent, and contained air throughout; the lower and posterior portions contained more blood than the upper—hypostatic congestion. In the lower portion of the trachea was some foamy liquid. The heart was well covered with fat; the heart substance and valves were nowhere abnormal. The right half of the heart was filled with dark fluid blood; the left was empty. The abdominal organs showed nothing remarkable; the omentum was normal. The contents of the stomach consisted of a greenish-yellow turbid fluid with single white flakes, and amounted to about 200 cubic centimeters. The mucous membrane of the stomach showed circumscribed areas of injection; in these areas the blood vessels were much developed and enlarged; here and there were small ecchymoses. The contents of the duodenum consisted of a thick, almost mushy, mass, which was much mixed with mucus; the color was cloudy yellow. The greenish color of the stomach contents was here less observable. The mucous membrane of the duodenum, and of the rest of the small intestines, showed the same hyperemic areas as the stomach. The contents beyond the duodenum were of a more solid consistence, and the lower portion of the small intestine and the whole large intestine contained feces of a yellow color and good consistence. In the small intestine several dead ascarides were found. Nothing remarkable was observed in the liver; yet the color of the contents of the gall bladder was dirty brownish. The spleen was perfectly normal in every way. Nothing abnormal could be discovered in the kidneys, ureters, or bladder. The latter was moderately filled with clear urine. The dura mater was normal; the pia mater at the sagittal suture showed opacities and patches. The brain was somewhat anemic, and the sinuses contained but little blood. For chemical analysis were reserved the stomach and contents, a portion of the œsophagus, a piece of the liver, a portion of the duodenum, contents of duodenum, and blood from the heart; also the pills found in the stocking. The stomach, part of the œsophagus, and portions of the liver and duodenum were analyzed together, and yielded a residue of digitalis. The contents of the stomach gave the reactions of digitalis,

and several very small green particles found in the stomach were reserved for the microscope. Digitalis was also present in the contents of the duodenum. Analysis of the blood gave negative results. Chemical and microscopical analyses of the pills showed them to consist of digitalis powder; the particles from the stomach were the same. The physiological test was also carried out, and with success.

The symptoms, which the deceased had suffered, are described in the report of the examination. He complained chiefly of loss of appetite, nausea, constipation, headache, and dizziness. On examination, the ill appearance and a strong smell from the mouth were very noticeable; the tongue was heavily coated; pressure over the stomach occasioned so much complaint of pain that there arose a suspicion of exaggeration. There was no fever. The pulse was slowed to 56 per minute. There were no other symptoms. In spite of appropriate diet and medicine his condition did not improve. On December 21st, the pulse was still slower, 52 per minute. On December 26th, he vomited, and, on emptying the vessel, greenish, slimy masses had been noticed; he was, therefore, given a vessel in which to preserve the vomit, in case of its happening again, for examination. This vessel was not used, and no vomiting had been noticed. His sick condition remained the same till the end of the month, and he lost more or less strength. He complained of darkness of vision, noises in the ears, and great weakness. Examination of the pupils showed that they reacted well and were of equal size. On January 9th, he complained of swelling in the neck and difficult deglutition. Examination showed no reason for the swelling, and the physician wished to make a further examination of the abdomen with the patient in an upright position; but the patient could not stand on account of faintness, which disappeared after a few minutes of rest. In the course of this day and the preceding he complained of hiccough. Early in the afternoon he wanted to go to stool; he had hardly arisen from his bed when he sank down, and in a few minutes was dead.

869.—Case XXI. Poisoning by Digitalin.

(Compiled by Assistant Professor Charles Harrington, M. D., of Harvard Medical School.)

(Tardieu, l'Empoisonnement, 1875, p. 809. Annales d'Hygiène Publique, 1864, Vol. II. p. 105.)

On November 17, 1863, Madame de Pauw died, under circumstances which excited suspicions of poisoning. These suspicions were directed against Dr. Couty de la Pommerais, who was pecuniarily interested in her death. The body was exhumed and examined

thirteen days after death. It was in a remarkably good state of preservation; it was well nourished and plump, and showed no evidence of disease. There were no marks of violence. The skull was intact; brain normal. Nothing remarkable about the mouth and throat. Lungs perfectly healthy; no hyperemia, no inflammatory changes, no tubercles. Heart normal, and contained considerable half-coagulated blood. No fluid in the peritoneal cavity. Liver, spleen, and kidneys normal. The stomach and intestines showed, in places, several suffusions of blood and hyperemic points; there was nothing pointing towards acute or chronic inflammation. The sexual organs showed no evidence of disease or violence. In the uterus was a fetus of seven or eight weeks.

The conclusions drawn from the autopsy were that there were no pathological changes to account for death from natural causes, and that the absence of any characteristic changes, and the appearances in the digestive tract, furnished reasonable grounds for suspecting death from poison, which could be proved only by chemical analysis. The stomach, intestine, lungs, heart, liver, spleen, and kidneys were reserved for analysis. The floor of the sleeping room of the deceased was also examined. Various substances found in the possession of the accused, and his books and papers, were investigated.

Among 900 chemical and pharmaceutical preparations (mostly homeopathic remedies) was a large quantity of deadly poisons, out of all proportion to the ordinary needs of a physician and more especially of a homeopath. There were four vials of white arsenic, three of corrosive sublimate, besides sulphate of copper, black hellebore to the amount of 125 grams, powdered nux vomica 250 grams, powdered stramonium 250 grams, powdered aconite 350 grams, powdered belladonna 250 grams, powdered digitalis 125 grams, prussic acid, muriate of morphin, strychnin, digitalin, etc., etc. It was shown that the accused had, on October 4, 1861, purchased 50 centigrams of digitalin; on June 11, 1863, 1 gram; June 19th, 2 grams; total 3.50 grams, of which but 0.150 (or less than one twentieth of the whole amount) was found. So large an amount of this poison to be used in that space of time is out of all proportion to the ordinary needs, not only of a physician, but even of a very busy apothecary, since the poison is of such a dangerous character that it can be given only in very small doses (1 milligram or less); 3.50 grams are, therefore, at least 3,500 doses.

Witnesses testified that the deceased had gone about her duties and had been in good health up to the evening before her death. The

first symptoms appeared in the night, and were repeated violent vomiting, and rapidly increasing weakness. The physician found her pale, very agitated, bathed in cold perspiration; she complained of intolerable headache; the pulse was irregular, intermittent, and hardly perceptible; the heart's action was tumultuous, irregular, and ceased from time to time.

On undertaking the chemical analysis, it was noticed that the stomach and intestine were not at all decomposed and gave off no odor; the other organs were undergoing decomposition. The latter were analyzed for mineral poisons, but with a negative result. The stomach and intestine were examined for organic poison. Various extracts of these organs were made and employed for physiological tests.

Scrapings from the floor where the deceased had vomited were analyzed; no mineral poisons were detected, and the alcoholic extract was further employed with the others above mentioned.

The first experiment was performed with the last-mentioned extract. A strong medium-sized dog was taken; incisions were made in the integument of the thigh, and 5 grams of the extract were introduced into the connective tissue; the wounds were then closed. Before the operation the heart-beats numbered 110 per minute. The dog remained apparently well during the first forty-five minutes. He then lay down and began to lick the wounds. At the end of two hours and twenty-five minutes he vomited three times, the vomitus being mucus and a little bile. He then lay down again very weak and anxious. His heart's pulsations were 94 per minute, irregular and intermittent, now ceasing, then tumultuous. Respiration quickened and somewhat intermittent. One hour later repeated vomiting; pulse 74. Seven hours from the beginning of the experiment the dog was quite exhausted; on being urged to move he would retch and vomit; pulse 68, and still more irregular and intermittent. Twelve hours later he was quite cold, but in full possession of consciousness; pulse 40, weak and very irregular. Respiration superficial, frequent, intermittent. Three hours later the dog died almost without any agony, and without any coma. Death occurred in twenty-two hours from the beginning of the experiment. On autopsy the organs were found to be normal; black, thick, partially coagulated blood was contained in the auricles and ventricles. The auricles were dilated, the ventricles contracted. Apex and vicinity bright red. Two grams of the extract mixed with water were introduced by means of a funnel into the stomach of a rabbit. The pulsations of the heart were

slowed, irregular and intermittent. Respiration embarrassed, and shortly before death intermittent. In two hours and five minutes the pulse was 41. Death occurred in two hours and forty-five minutes. On autopsy the organs, excepting the heart, were found normal. The ventricles were contracted, auricles dilated; apex bright red. The heart wall showed several abnormal, small, raised, red spots.

Five grams of the mixed extracts from the stomach and intestine were introduced into the thigh of a strong medium-sized dog. The pulse was 102 before the operation. In two and a half hours the animal was depressed and anxious, and lay down. Respiration was stertorous and intermittent; pulse 84, irregularly intermittent. Vomited twice. In six hours, pulse 55; respiration superficial and labored. He was restless and uttered faint cries; fully conscious. Twelve hours later, pulse 70, dog much better. In twenty-four hours from the beginning of the experiment the pulse was 90, and still somewhat intermittent. Perfect recovery followed.

Experiments made with an alcoholic extract from scrapings from the floor, where there was no vomitus, were without result. The extracts from the floor differed chemically from each other. That containing some of the vomitus was brown in color, had a rancid, oily odor, and a bitter taste. Tannic acid threw down a precipitate; sulphuric acid produced a purple-red color; hydrochloric a green. The other was not bitter, and did not react with acids as did the first. Attempts to separate the active principle of the poison from the extracts were fruitless.

The preceding experiments showed that the poison was one having a special action on the heart. They were repeated with frogs. The hearts of three frogs were exposed in order to enable observation of the action. No poison was given to the first frog; the second received 6 drops of a solution of digitalin (0.010 gm. digitalin in 5.00 gm. water) under the skin; the third was given 0.050 gram of the poisonous extract from the floor. The following table shows the comparative rapidity and rhythm of the heart's contractions:—

		Frog No. I.	No. II.	No. III.
After	6 minutes	42	20	26
	10 "	40	16 irregular	24 irregular
	20 "	40	15	20
	28 "	38	0	12 very irregular.
	31 "	36	0	0

The hearts of the two poisoned frogs showed contracted ventricles

and dilated auricles. The heart muscles showed no microscopic changes. This experiment was repeated several times, and the slowing of the heart and the irregularity of the rhythm were constant phenomena. Notwithstanding the amplitude of the slowed heart beats, the blood was, toward the end, not completely ejected. These symptoms, and the condition of the heart after death, are in full agreement with the characteristic phenomena of digitalis poisoning.

The results of these experiments, the lack of abnormal post-mortem appearances, the previous good health of the woman, the finding of forged letters proving the accused to have anticipated the death of the deceased, the possession and use of such a quantity of the deadly poison by the accused, and the fact that he was pecuniarily interested in the woman's death,—made a very strong case against him. He was declared guilty, and subsequently executed.

870.—Case XXII. A Peculiar Case of Poisoning by Carbon Monoxid.

(Compiled by Assistant Professor Charles Harrington, M. D., of Harvard Medical School.)

(Vierteljahrsschrift für gericht. Med. und öffentl. Sanitätswesen, Bd. XXIX. p. 272.)

On September 27, 187--, towards 5:30 o'clock in the afternoon, two workmen, C. H. and W., were engaged in a paper factory filling a large iron kettle with rags. The kettle was of spherical form and 2.40 meters in diameter; it was in a large airy room with a number of others of the same sort. The kettles rotate on an axis, and, at the upper part of each, is an opening just large enough for a man to go through into the interior. The kettles are filled by means of a hopper arrangement coming through a trap door in the floor above, which is about 0.75 meter from the uppermost surface of the kettles. The partially cleansed and finely cut rags are fed loosely from the hopper into the kettle, after which a man goes through the opening and treads them down more firmly. The amount necessary to fill a kettle varies according to the character of the rags; from 1,000 to 1,500 kilograms. Slaked lime in the form of milk of lime is then introduced through a tube; the amount used varies according to the rags, and requires from 100 to 200 kilograms of calcined lime for its preparation. The opening is then closed and the kettle rotated by machinery; steam is introduced through a pipe in the axis and the resulting lye runs off through an escape pipe. The plugging of these pipes by rags is prevented by sieves. The operation requires for completion from four to twelve hours.

When the workmen had filled the kettle with rags, C. H. went inside in order to tread them down; meanwhile, W. left the factory to do an errand. On returning after a short interval he missed his comrade; receiving no reply on calling out, and finding no reason for his absence from the room, he climbed into the kettle. There he noticed that the rags were more firmly packed, filling only one half of the kettle; lying on the rags was the apparently lifeless H. After vainly trying to rouse him, he climbed out without having felt in the least unwell, brought a lamp, and called another workman, J. H., to his aid. W. again climbed in while J. H. held the lamp at the opening in order to light up the interior. It was then about 6 o'clock. When W. had convinced himself that H. was dead, he went to give information at the office. Meanwhile, several workmen came into the room, and the workman J. H., a very strong man, climbed into the kettle and lifted the corpse, so that the others standing above could draw it out. After about half an hour, during which efforts to revive the deceased were fruitless, W. again climbed into the kettle to continue the packing. To his horror he found a second corpse, that of J. H. who had lifted out the first, and who, in the general excitement had not been missed. The death of C. H. had not been ascribed to the development of gas, but to apoplexy, and that J. H. might require assistance in getting out of the kettle had not been considered. W., while trying to revive J. H., became unwell, and was barely able to get out of the kettle. The symptoms were those of suffocation, and disappeared only after long exposure to fresh air.

This led to the conclusion that poisonous gas must have been developed in the kettle, and caused the two deaths. In order to remove the second corpse with more ease and less danger, the kettle was turned on its axis till the opening came nearer the floor. The overseer then introduced a burning lamp and the upper portion of his body into the cavity, to discover the whereabouts of the corpse; on finding it, he withdrew, and instructed the workmen as to its position. A workman now introduced his arm into the opening up to the shoulder, for the purpose of reaching and drawing forth the body. A second man stood next to him with one hand inside to seize the body as soon as it should be brought within reach; a third stood by ready to assist. The overseer stood with a lamp about three yards from the hole, when, suddenly, a violent explosion occurred, more or less severely injuring the three workmen,—especially the first mentioned. The contents of the kettle were ignited, but soon quenched by means of a powerful stream of water, then removed from the vessel.

Examination of the wet and partially charred rags revealed the presence of a large amount of fragments of lime. This gave an important hint towards explaining the cause of death, which, on the supposition that C. H. had performed the operation in the usual manner, was very mysterious. The theory of the overseer was that C. H., in order to avoid slaking lime, and thus save an hour, had thrown in the lime before filling with rags, intending to let the steam slake it. The workmen W. had noticed nothing of the sort, yet it had been possible for H. to throw in lime while W. brought rags. The director admitted it to be very possible that a certain amount of water had been left in the kettle from the last cleaning.

The theory of the development of carbonic oxid and hydrid from dry distillation of the rags was strengthened at the autopsy by the very noticeable characteristic light red blotches (suggillations) on the body.

The autopsy was performed 62 hours after death. The same bright red color was observed in the interior of the body, especially marked in the blood and muscles. This red color was not so marked in the body of J. H. Decomposition was comparatively far advanced in both bodies; rigor mortis was present in J. H. only in the lower extremities. In both cases, vomitus was found in the respiratory tract; in the body of C. H., only a small number of doughy clumps were found in the foamy mucus which filled the trachea, but larger masses were present in the mouth, nose, and œsophagus. In the lungs of J. H., as far as the medium-sized bronchi, fine food particles and foam were discovered; the trachea contained more of the same, and, immediately above the bifurcation, was completely plugged by a strongly wedged mass of food. The lungs of both were, as is usual in suffocation, edematous, and on the surface emphysematous. No ecchymoses were found in the lungs, heart, or pericardium. The blood vessels of the skull and brain of C. H. were very full; those of J. H. not in so marked a degree. The spectroscopic examination of the blood was, unfortunately, not carried out.

The investigation of this case included consideration of the nature of the poisonous gas. It was already evident that, when inhaled, it was either incapable of supporting life, or capable of exercising an active injurious influence, or both, and that it was an explosive. As to its development: Admitting the presence of a certain amount of water in the kettle, which, if not left from the previous cleaning, may have been present as dampness in the rags (hygroscopic, etc), it is evident that, when brought into contact with the unslaked lime, hy-

drate of calcium will be formed, with the development of a great amount of heat. The rags in contact with the lime would thus be subjected to a sort of dry distillation, or, owing to a lack of oxygen, to an incomplete combustion. Under such circumstances, gaseous compounds would be developed, of which carbon monoxid and marsh gas are the most important. Both of these, and especially the former, are specifically lighter than air, with which they easily mix; both are colorless and odorless; both may be inhaled without any irritating effect on the mucous membrane. The marsh gas of itself produces no injurious effects when inhaled, though it is incapable of supporting life. Mixed in certain proportion with air, and ignited, it explodes violently; the explosion does not occur when the proportion of air is less than six times, or more than fourteen times, that of the gas. The carbon monoxid, on the contrary, is not only incapable of supporting life, but acts as a poison on respiration. Mixed with air in a proportion of 5 per cent, it soon brings on unconsciousness and death. Neither in the pure state nor when mixed with air is it explosive; it burns in the air with a blue flame to carbon dioxid. The explosion which occurred in this case is easily explained, and the reason of its nonoccurrence on the first exposure of the lamp to the atmosphere of the kettle is, evidently, that the proportion of marsh gas in the kettle was too small or too large for an explosion to occur. The post-mortem appearances—especially those of C. H.—were characteristic of poisoning by carbon monoxid. The bright red color and the fluidity of the blood in both cases, the bright red color of the suggillations on the body of C. H., and the bright red muscles of both, in spite of advanced decomposition, have been mentioned. The character of the blood is especially characteristic of poisoning by carbonic oxid. The body of J. H. was not so marked as that of C. H., nor was the blood so light in color; decomposition was further advanced. The relatively few characteristic appearances of carbonic oxid poisoning in the case of J. H., and the fact of the trachea having been firmly plugged by vomited food, had an important bearing on the verdict of the examiners.

The finding was in substance as follows:—

1. The death of C. H. was caused by inhalation of carbonic oxid gas developed in the rags by the heat produced by the change of quicklime to milk of lime.

2. The death of J. H. was due, directly, to inhalation of vomited food, and, indirectly, to poisoning by carbonic oxid gas.

3. The explosion in the factory was due to the ignition of a mixture of marsh gas (light carbureted hydrogen) and air.

The points of peculiar interest in the above case are, first, the short duration, and second, the manner in which the gas was developed.

871.—Case XXIII.—Poisoning by Gelsemium.

(Compiled by Assistant Professor Charles Harrington, M. D., of Harvard Medical School.)

(American Journal of Pharmacy, January, 1870, p. 14.)

On the 30th of January, 1869, three teaspoonfuls of fluid extract of gelsemium were administered to a young, healthy married woman, several weeks advanced in pregnancy, who, at the time, complained of no serious illness. In two hours after taking the dose, the patient complained of pain in the stomach, nausea, and dimness of vision. These symptoms were soon succeeded by great restlessness, ineffectual efforts to vomit, and free perspiration over the body. At the expiration of about five hours the pulse was found feeble, irregular, and with intermittent beats; there was great prostration, with irregular breathing and slow respiration. The skin was dry; extremities cold; the pupils expanded and insensible to light; the eyes fixed; inability to raise the eyelids. The vital powers rapidly gave way, and, without convulsions, death occurred in about seven hours and a half after the poison had been taken.

Post-mortem appearances.—Eight days after death the body presented the following appearances, as described by Dr. J. H. Stephenson, who made the autopsy: Countenance natural as in sleep. No emaciation, and body in a perfect state of preservation. Cadaveric rigidity very slight. The back of the neck and between the shoulders, extending the full length of the spine, as also the depending parts of the thighs, and the arms to the elbows, presented a congested appearance. The membranes and substances of the brain and medulla oblongata were normal. The adipose tissue remarkably thick, and highly tinged throughout with bilious matter. Lungs slightly collapsed, natural in appearance, and superficial veins congested. Heart normal in size, superficial veins injected, and the cavities greatly distended with dark grumous blood, inside of which was found a well-defined membrane identical in appearance with that found in diphtheria and pseudo-membranous croup. The abdomen presented no tympanitic distention. Stomach was slightly distended with gas, and contained a small quantity of ingesta. Peritoneum and intestines in a healthy condition. Liver and investing membrane normal; left kidney congested. The uterus was slightly enlarged and contained a fetus of about five weeks' devolopment.

A small quantity of the contents of the stomach, which escaped from the organ at the time of the dissection, was collected separately in a small bottle, the stomach with the balance of its contents was placed in a larger bottle. These bottles, with their contents, were carefully sealed and remained undisturbed until the 17th of May. At this time the contents of the bottle containing the stomach were found to have undergone considerable decomposition. A little pure alcohol was added to the decomposing mass, and it was then allowed to remain until the 13th of June, when the chemical examination of the contents of both bottles was commenced. On analysis of their contents by Dr. T. G. Wormley, both gelsemic acid and the base gelsemin were found.

It will be observed that, in this case, only three teaspoonfuls of the fluid extract were taken. Presuming it to have had about the same strength as a preparation examined by Dr. Wormley, the quantity of the alkaloid contained in this amount could not have much exceeded the sixth part of a grain. This would seem to indicate the alkaloid to be one of the most potent poisons at present known. On comparing the intensities of the reactions of the several reagents applied to the base recovered from the stomach, with those obtained by the same reagents from solutions of the alkaloid of known strength, it was inferred that the quantity of the base recovered in this case did not much, if any, exceed the fiftieth part of a grain.

The fact that the stomach with its contents had undergone considerable decomposition, and also that the chemical examination was not made until some months after death, would seem to indicate that the poison is not readily destroyed by decomposition, and that it may be recovered after comparatively long periods, even when taken only in small quantity.

872.—Case XXIV. Poisoning by Belladonna.

(Compiled by Assistant Professor Charles Harrington, M. D., of Harvard Medical School.)

(The Lancet, July 16, 1870, p. 83.)

Ann H., aged sixty-six, swallowed about a teaspoonful of belladonna liniment on March 19, at 11 A. M. At 11:30, a friend found her wandering in her mind, and, having ascertained what had happened, administered an emetic, but without inducing vomiting; subsequently two other emetics were given, but all failed to have any effect. At 2 P. M. she was taken to the Royal Infirmary, Bristol; she was then delirious, talking fast, and throwing her arms about in an excited but feeble way; her pupils were widely dilated and insen-

sible to light; her pulse was 126. The stomach pump was used, but very little fluid was brought up. At 2:45 she was very restless, tossing about in the bed, picking at the bedclothes, and throwing about her arms in a meaningless way, but partially conscious of what was said to her. She wished to sit up in bed. She could swallow fluids with difficulty, and drank some strong coffee. Pulse 116; respiration natural, rather slow. She was ordered a draught containing 20 minims of tincture of opium, and a hypodermic injection of acetate of morphin (⅓ gr.) was given soon afterwards. A "calabarized disk" was placed in one eye (the left); in about twenty minutes the left pupil had contracted to a pin's point, the right pupil remaining widely dilated. At 3:30 she was less active in her movements, and seemed more drowsy. The morphin injection (¼ gr.) was repeated. At 4:30 the right pupil was still widely dilated, the left remaining contracted. Her pulse was 100, of good strength; breathing slow. She was more comatose, and breathed stertorously if undisturbed for a few seconds. Another subcutaneous injection of acetate of morphin (⅓ gr.) was given. At 6:15 she was deeply comatose, and apparently had ceased to breathe; no respiration was visible for a full minute, but her pulse was moderately full, at 100; her hands were blue, and feet tending to get cold. Artificial respiration by Silvester's method was resorted to, when she began to breathe again regularly, but very slowly, about four respirations occurring in a minute. Enemata containing ammonia, brandy, and coffee were given, and galvanism to the respiratory muscles was resorted to. At 8:30 she remained deeply comatose; no reflex movements could be obtained; respirations were five or six per minute; pulse 100; cervical veins were much distended; lips were blue, and the heart's impulse diffused and weak. Brandy and coffee enemata were given every hour, and galvanism was used at intervals. At 1:30 A. M., on the 20th, respirations were six or seven per minute; pulse 100, but variable, at one minute good, at another very weak and small. Venous distention in the neck had subsided. The right pupil remained dilated widely and insensible to light, and the left contracted till the time of her death, which took place at 3 o'clock, sixteen hours from the time of her having taken the poison. At the post-mortem examination, thirty-four hours after death, the lungs were found full of blood; the right side of the heart contained but little black blood, the left side being very firmly contracted. The brain was slightly congested. The stomach and other organs presented nothing abnormal. At the time of the autopsy both pupils were equally dilated.

The patient took, in divided doses, 11/12 of a grain of acetate of morphin subcutaneously, and 40 minims of tincture of opium by the mouth. Not the slightest effect was noticeable in the right pupil, *i. e.,* the one which was not acted on by the Calabar bean. Excepting that this pupil was dilated, the patient's condition exactly resembled that of opium poisoning, after the delirium had subsided into coma. No evident result was traceable to the morphin administered, except that the coma deepened, and the breathing got slower; but these symptoms doubtless were due to the belladonna, uninfluenced by the medicinal doses of opium and morphin.

873.—Case XXV. Poisoning by Atropin.

(Compiled by Assistant Professor Charles Harrington, M. D., of Harvard Medical School.)

(British Medical Journal, Oct. 5, 1878, p. 516.)

Charles S., aged forty-five, a gardener, swallowed a teaspoonful of liquor atropiæ sulphatis on August 6, 1875, and was taken to the Fulhane Hospital. He said that he was attending an ophthalmic hospital for an affection of the eye, and received some drops, as well as a larger bottle of medicine for internal use. He was in the habit of pouring out his daily allowance of the latter into an ounce and a half vial, and carrying it in the same pocket with the atropin. About 1 P. M. he left work, and, when he took his medicine, by mistake, drank from the wrong bottle. He felt nothing particular, but told the clerk, who brought him home, what he had done. He was then quite sensible; there were no symptoms or signs whatever except excessive nervousness. The left pupil was dilated from a previous application of the atropin. Mr. Greenway gave him an emetic of ipecacuanha wine; but this not acting quickly enough, it was followed by 20 grains of sulphate of zinc in mustard and water, and he was told to walk about. At 1:39 P. M. his pulse was 140, small and regular; temperature 98°. He staggered as he walked, and complained of giddiness. The pupils were not affected. No vomiting supervening, his stomach was washed out with the stomach pump. He lost power over his legs, and became quite helpless and unconscious; his pulse became excessively weak and irregular; he was given 1 dram of tincture of digitalis in brandy. At 2:15 he began to talk quickly, and looked flushed. At 2:45 the pulse was 148, full; the skin was dry. The face was very much flushed. He had very violent delirium, requiring three men to hold him. The pupils had

gradually enlarged till they were nearly fully dilated. The tongue was dry and brown; he could swallow after some compulsion. Vomiting continued at intervals, and black coffee was given him now and then. His feet became cold; mustard was applied to them. At 3:15 P. M. the pulse was 156, smaller; temperature 99°. He was quieter, and had a tendency to sleep. The pupils were the same, and insensible to light. He did not retract his legs when they were pricked or pinched. When spoken to, he did not seem to hear, being evidently quite unconscious. At 4 P. M. the pulse was 144. He seemed generally the same, except that when his name was shouted in his ear, he opened his eyes and turned his head in the direction of the sound. For the first time the respiration seemed labored and quick, accompanied by flapping of the cheeks when he was permitted to sleep. He now moved both arms and legs, at times; twitching of some of the muscles was noticed. He had a mustard poultice over his cardiac region, and small quantities were administered. An enema of castor oil was given. At 7 P. M. the pulse was 140; respiration 40 per minute. The tongue was as before, the face less flushed, and the skin still quite dry. He was decidedly more conscious, as, when told to put out his tongue, he did so, and said "yes" when called. He caught at imaginary objects. On account of his precarious state, Dr. Milner Fothergill was requested to see him. He advised that the treatment be continued.

Dr. Fothergill spoke to him, and he answered fairly intelligibly. At 9:15 P. M. the pulse was 140, small and regular; temperature 102°; respiration 50 per minute, laboring. On cursory examination of the chest, a few moist *râles* were heard about the bases. He recognized some friends who came to see him. The skin and tongue were as before. As soon as he fell asleep he commenced to talk unintelligibly. The pupils were as before. Neither bowels nor bladder had acted. At 10:35 P. M. the pulse was 140, full and regular; temperature 102.4°; respiration 60. The bowels had acted slightly. He was inclined to become violently delirious, as Dr. Fothergill had foretold. His general condition was unchanged. He had been having eggs, coffee, milk, etc., and a little brandy at intervals. At 11:45 the catheter was passed, but the bladder was empty, and there was no dullness over the pubes. He had considerable tympanites, and tenderness over the abdomen.

August 7, 1:30 P. M. The pulse was 132, full and regular; temperature 103°; respiration 56. He passed a fair amount of urine in the sheets. His skin, pupils, and tongue were as before reported. He complained of great thirst, to allay which ice was given. At

2:45 the pulse was 130, full and regular; temperature 102.6°; respiration 48. He was now nearly conscious, and intelligible in his answers; but directly he fell asleep, he commenced to talk and mutter. At 4:30 the pulse was 92, small and slightly irregular. He now remembered taking the poison, and knew where he was. He looked very pale. At 6:30 the pulse was 115; temperature 100.4°; respiration 30. The bowels had acted very freely three times. Ammonia and bark were ordered. At 11:45 the pulse was 140, full and regular; temperature 102.2°; respiration 36. He complained of soreness at the pit of the stomach. The bowels acted twice again. He had perspired for the first time. The tongue and mouth were less dry. On applying the light of a candle to the pupils, the right contracted slightly; the left did not. He said he had had very pleasant dreams. At 9 P. M. the pulse was 144, full; temperature 102.4°; respiration 52. The skin was now moist. He passed some urine of an amber color, specific gravity 1.015; no albumin. At 10:40 P. M. the pulse was 156, small; temperature 100.6°; respiration 52. The pupils were still much dilated. He complained of no pain.

August 8, 10 A. M. He passed a fair night, sleeping at intervals. He vomited once; the bowels acted four times. He took his food well. He complained of some pain over the right side of his chest, and had commenced to cough and expectorate. He was very thirsty. The tongue was as before. The left pupil still dilated; the right was much smaller, and contracted on looking at a bright light. On examining the chest in front, both lungs were resonant and otherwise normal. Behind, at the base of the left lung, a limited area of crepitation was found, but no dullness. Over the right base was a small crepitation extending upwards to the spine of the scapula, and very slight dullness. The sputum was rust-colored and scanty. He was ordered a mustard poultice to the right base, and 3 grains of carbonate of ammonium in decoction of senega every three hours; also 2 drams of brandy every hour, with milk, beef-tea, etc.

August 9, 10 A. M. Pulse 160, regular; temperature 100.2°; respiration 60. He slept fairly well during the night, but in the morning complained again of pain in the right side of the chest. On examination of the chest, dullness as far as the spine of the right scapula was found, and tubular breathing and bronchophony, but no crepitation. Over the left lung, posteriorly, there was rough breathing, but otherwise the respiration sounds were normal. At 11 P. M. a sudden change for the worse set in, and the symptoms increased until death, which occurred at 2:10 A. M.

The autopsy was performed sixty-five hours after death. The body was well nourished. The abdomen was swollen, tympanitic, and had a green color. The right side of the chest was duller than the left. There was very little hypostatic congestion. The pupils were slightly dilated. There was no rigor mortis. The pericardium contained no fluid. The heart was enlarged and very soft; the right half was much distended with dark coagulated blood; the coagula extended into the venæ cavæ. The left side of the heart was empty, and the muscular walls were soft. The mitral valve admitted three fingers. The pulmonary valves were competent and healthy. The aorta was slightly incompetent and atheromatous.

The middle lobe of the right lung was healthy and crepitant throughout; the lower lobe was hepatized and noncrepitant. There was slight exudation of blood-stained material, on section. There was no pleuritic effusion or adhesion at the base. The liver was natural in size, extremely soft, somewhat pale in color, and fatty on section. The spleen was about five inches long, very soft and friable. The kidneys were also very soft, breaking down on removal. The cortex was congested, and the capsules very adherent. The bladder contained a small amount of urine. The œsophagus had a patch of congestion at the upper extremity, and, at the cardiac end, some congestion extending into the cardiac end of the stomach, which was also congested as far as its middle. The duodenum was congested at various points. At the lower end of the ileum there was a patch of redness of the size of the palm of the hand. No breach of surface existed. The pia mater was excessively congested. The brain, on section, appeared to have more bloody points than usual; the lateral ventricles contained some bloody serum. The substance of the brain was very soft.

874.—Case XXVI. Poisoning by Strychnin.

(Compiled by Assistant Professor Charles Harrington, M. D., of Harvard Medical School.)

(From reports of the trial of David L. Magoon.)

Mrs. D. L. Magoon, of Raymond, N. H., a woman of advanced years, died suddenly in the afternoon of August 31st, 1874. Her husband, David L. Magoon, on that day took from his pocket a bottle of "essence of peppermint," poured some out into a tumbler, added hot water, and carried it to her in her bedroom where she lay sick. On account of its extreme bitterness she was unable to drink the whole, and he threw the remainder away. In less than an hour she was dead. She had frequently complained of cold and shivering, and at these times, everything tasted bitter.

On her death, nothing peculiar was noticed about the position of the body. The face wore a peaceful expression; the hands were clenched unusually tight; the body was so stiff that the clothes had to be cut off.

A little less than a year later (August 12th, 1875), the body was exhumed, on account of suspicions of poisoning due to remarks dropped by the husband, and to the finding of a strychnin bottle in his pillow case; an examination was, therefore, made. The organs were found in a good state of preservation; the stomach was remarkably well preserved. The latter organ contained ½ ounce of fluid; it was, with its contents, submitted to Dr. Hayes, of Boston, and Dr. E. S. Wood, of the Harvard Medical School, for chemical analysis. On September 3, 1875, the body was again exhumed, and a third of the liver and a fourth of the intestines were removed and submitted for analysis. The stomach was found to contain 0.15 grain of strychnin; the portion of liver contained 0.23 grain; the poison was detected in the intestines, but it was not estimated.

The prisoner confessed his guilt after a disagreement of the jury, and died, probably by suicide, before the time appointed for a second trial.

875.—Case XXVII. Poisoning by Strychnin.

(Compiled by Assistant Professor Charles Harrington, M. D., of Harvard Medical School.)

(From the report of the trial of Edwin W. Major.)

Ida Major, of Wilton Centre, N. H., a young woman eighteen years of age, the mother of four children, and about to bear a fifth, died, on December 20th, 1874, under very suspicious circumstances. On that day between 4 and 5 o'clock she had eaten supper, after which she went to bed; her husband, Edwin W. Major, gave her some "medicine," which, he explained, would taste very bitter. Shortly before 7 o'clock, her husband called in two of the neighbors, who found her complaining of violent headache, blurring of the eyes, and dry mouth; she had had convulsions at 5:30, and, shortly after their arrival, she was again seized with the spasms. During the spasms she frothed at the mouth, and uttered shrieks, but was perfectly conscious. The convulsions continued until 7:20 P. M. when she died. She spoke fifteen minutes before death. On laying out the body, the feet were found to be so contracted that the stockings were with difficulty removed; the arms could not be straightened, and the chemise had to be torn off.

The autopsy was performed two days afterwards. The face was lifelike and florid; body very stiff; feet arched and turned outwards. Flexor muscles of arms contracted like those of lower limbs, but not to such a marked degree. Brain normal; the veins were congested. Lungs, heart, kidneys, bladder, pancreas, gall bladder, and intestines were normal. Pregnancy was far advanced; labor had apparently commenced. The external surface of the stomach was normal in appearance, except that the blood vessels—especially the veins—were engorged. The mucous membrane was covered with mucus; the vessels—especially along the lesser curvature—were filled with blood. There was no loss of substance of the mucous membrane. The congestion was most marked upon the lesser curvature near the cardiac end, and at the pylorus; the blood was normal in color, being red in the arteries and dark in the veins. The contents of the stomach amounted to 4 ounces, and contained potato starch, oil globules, vegetable fiber, amorphous matter, and sarcinæ. The organ and its contents were sent to Dr. E. S. Wood, of the Harvard Medical School, for chemical analysis. Twenty days later the body was exhumed and the liver removed for analysis. . The lower extremities were still as stiff as on the day of the autopsy; the arms were somewhat relaxed. The upper part of the spinal cord was exposed; it was normal, but the blood vessels were engorged.

The chemical analysis detected 1.72 grains of strychnin in the contents of the stomach, 0.51 in the stomach, and 0.23 in the liver.

The guilt of the accused was proved at the trial, and he was subsequently executed.

876. Case XXVIII. The Trial of Mrs. Marcus M. Rogers for the Murder of Her Husband for the Purpose of Obtaining an Insurance of $500 on His Life.

(The notes of this case were given the writer by the medical expert, Dr. Daly.)

This case was tried before the county court at Bennington, Vermont, early in December, 1903. A poor attempt was made by the accused to prove that her husband committed suicide by the pinning of a note on his hat, supposed to have been written by him, stating that he had drowned himself because he was tired of life. The defense claimed, also, that his death was caused by a blow on the frontal bone. "The murdered man had a contusion over the right eyebrow, or exactly occupying a portion of the eyebrow. It presented no external discoloration, and was less than an inch in length, and it

did not extend through to the supraorbital ridge." On dissecting the scalp, Dr. Daly, who performed the autopsy, examined the under side of the contusion, and "found an ecchymosis, oval in shape, of an inch and a quarter in its long diameter. There was no discoloration of the periosteum, the ecchymosis was confined to the under side of the skin, and this was directly under the external contusion."

"The lungs presented some of the features of death by drowning. There were, on section of the lungs, appearances of frothy mucus; but there were no ecchymotic taches in the trachea, and no water or foreign substance in the air passages below the epiglottis. This absence of foreign matters was almost conclusive to the mind of the examiner, because the water in which the body was found was literally impregnated with fine sawdust in a half-water-logged condition; it seemed impossible that he could have breathed after he had been immersed."

The evidence at the trial went to prove that his wife saturated a handkerchief with chloroform, which she held tightly to his mouth, and Dr. Daly expressed his opinion that the murdered man was thereby prevented from breathing rather than killed by chloroform intoxication. The victim had submitted, in sport, to allow himself to be bound with a rope so that he was unable to help himself against the force applied. "The trick was done at the river's brink. The heart and stomach were normal; the stomach contained a few ounces of partially digested food fluids. He had eaten his supper about four or five hours before his death."

The accused woman was convicted and sentenced to death. Her accomplice, who was present and helped her in the murder, was convicted and sentenced to life imprisonment.

877.—Case XXIX. Thayer Life Insurance Case. A Supposed Suicide. (May, 1903.)

Thayer resided on Walnut St., Newtonville, Massachusetts, and, up to about a year ago, was employed as a clerk in a large insurance office.

It appears that Thayer was arrested by a police inspector, May 27, 1903, charged with uttering a forged mortgage for $5,000 (on George I. Robinson, Jr., a Sears building broker).

When the case was called in court, it was continued until April 12th. He passed the night of April 30th at the Hotel Lenox, Boston, in bed with a constable, who was sleeping beside him. The next

morning he was found dead in this bed. He had been ailing, meanwhile, and had been attended by friends and relatives.

Medical Examiner F. W. Draper certified nephritis, complicated with a cardiac affection and congestion of the lungs, as the cause of death, after he had performed an autopsy upon his body.

Suspicion arising on the part of some of the life insurance companies that he had committed suicide, an order for exhumation was issued from the district attorney (Stevens). The investigation into the circumstances of Thayer's death became a case in the criminal courts of Suffolk county, on the ground that, by the common law, suicide is a crime against the state, as it deprives the commonwealth of one capable of performing military duty.

New York makes suicide a criminal offense; but, as in Massachusetts, it is held as being against the common law. An investigation into the causes of a suicide is made a matter for the district attorney's office.

The statutes of Massachusetts make it compulsory for boards of health to act upon the order of the district attorney for exhumation, it not being optional on their part; and Newton cemetery likewise is legally compelled to obey orders for exhumation from the board of health. The body was exhumed under the supervision of Medical Examiner Draper, and portions of the organs desired were turned over to Prof. W. B. Hills, Prof. Charles Harrington, and Prof. E. S. Wood, all connected with the Harvard Medical School, and recognized as chemists of high standing.

Prof. Hills and Prof. Harrington reported that they found no poison in these organs. The report of Prof. Wood to the district attorney was made in the following letter:

"Boston, Mass., July 17, 1903.

"I have made a very exhaustive analysis of the organs of Edwin M. Thayer, which I received from Dr. Draper on May 30th. These organs were said to be ⅓ of the liver, ⅓ of the stomach, ⅓ of the heart, and ⅓ of one kidney. I have analyzed these organs to see if they contained any prussic acid or cyanid of potassium, chloral hydrate, morphin, and any other narcotic poisons. I have been unable to obtain any positive proof of the presence of any of these poisons in the above-mentioned organs. I feel, however, that it is much to be regretted that the contents of the stomach were lost at the time of the autopsy, since the chemist relies chiefly upon the analysis of the contents of the stomach for the detection of certain poisons, and especially of prussic acid (or cyanid of potassium) and

chloral hydrate. Therefore, the failure to detect certain of the above poisons in the material received by me for analysis in this case does not prove with certainty that death was not caused by one of these poisons.

"Edward S. Wood."

878.—Case XXX. Trial of George C. Hersey for the Murder of Betsy Tirrell before the Supreme Judicial Court of Massachusetts.

(Boston, A. Williams & Co., 100 Washington St., 1862, by James M. W. Yerrington, Court Stenographer.)

Hon. George T. Bigelow, C. J.

Hon. Charles A. Dewey, Hon. Pliny Merrick, Hon. Reuben A. Chapman, Associate Justices.

The charge set forth by the counsel for the government was to the effect that the accused was engaged to be married to an older sister of the deceased, by name Mary, who died very suddenly during the month of January, 1860, after an illness of scarcely one week. While an inmate of the Tirrell family, the accused became very intimate with Betsy Tirrell, and, as afterwards discovered, seduced her, and she became pregnant by him. She apparently was aware of this during the month of April in the same year, and was much concerned thereby. It was brought out in the evidence that she had earnestly urged the accused to get her some drug, by taking which she hoped to get rid of the fetus before any discovery was made of her condition. It appeared during the trial of the case that the accused was not to be satisfied with this solution of the embarrassment, and conceived a design to accomplish her death by poison, with the hope of hiding his own as well as her disgrace. He therefore procured some strychnin, and administered this poison (then only having been known in medicine as a poison for fifteen years) to her in a spoonful of some unknown sweet preserve. Whether the victim knew that she was taking a fatal poison, or some drug which she supposed was only an abortifacient, does not appear in the evidence before the court; it would seem, however, from the sudden change of her manner, which had previously been that of a despondent woman, to a more cheerful and contented demeanor, that she was not aware of the kind of drug, nor that it was a deadly poison. She died within an hour after the suspected time when she had swallowed the poison; her young sister, who was sleeping by her

side, was suddenly awakened, and found the victim in convulsions. The victim had time before her death only to say, "I shall die, I shall die. Give me some physic." Very soon after, she ceased to breathe.

As two daughters had died from some unaccountable cause, who had, apparently, been in previous sound health, the family were alarmed and desired an autopsy to discover, if possible, whether the present daughter had died from some unnatural cause. The post-mortem examination showed that she was pregnant, and suspicion was attached to Hersey as being the father of the child, and her seducer. A coroner's inquest was ordered, and only the contents of the stomach were submitted to an analysis to search for some poison.

Examination of the medical evidence, which was proved satisfactorily to the jury, before whom the evidence was presented, showed that the accused was guilty of a deliberate murder of his victim, and a verdict to this effect was rendered. This evidence has certain points of interest to the medical expert of this later day. Here was an alkaloid used deliberately as a poison, with which an unlearned man, not a physician or medical student even, had caused a murder, and had probably selected this, as one which the medical faculty would have difficulty in discovering in the body after death. Fortunately for the ends of justice, the chemical character of this poison had been worked out carefully within the preceding seven years, so that it could quite readily be separated from the dead body. The physiological action of this poison had also been carefully studied, so that the symptoms of strychnin poison could be easily recognized before death; moreover, the symptoms were so distinct during the period which preceded death that their description by a person, unfamiliar with the effects of strychnin upon animal life, could present the portrait of its action in such language that a medical man ought to be able to recognize the poisonous agent. A notable character of these symptoms should be mentioned, *viz.*, the continuous or clonic form of muscular spasm, so well described by a witness at this trial, Prof. E. H. Clarke of the Harvard Medical School, who had remarked, in his course of lectures on the action of this poison, that it was one which the physician should be able to recognize as so different from the convulsive spasm known in natural diseases; the nonprofessional bystander, on the other hand, simply describes it as fits. The other physicians, also, were able, in this case, to explain to the court the peculiarity of these convulsions, as showing that they were the effect of a poison rather than the consequence of an ordinary disease. Again, we observe another important symp-

tom, as described by the physicians who performed the autopsy or were witnesses of it, *viz.*, the symptom of asphyxia, as shown by the emptying of the left auricle and ventricle of the heart, and the fullness of the right ventricle and auricle, with suffusion of the smaller blood vessels in the lungs and brain, also, the expression on the countenance of the dead body:—"I called the attention of the gentlemen to the drawing up of the angles of the mouth, exposing the teeth. As the body lay, the angle of the mouth was drawn up strongly towards the eyes, giving a grin to the countenance, and exposing the teeth. The fingers were strongly curved towards the palm of the hands. The body was quite rigid, when we examined it at the beginning of the afternoon, so that, when the arms were separated from the breasts, the elbow joints required to be forcibly broken down in order to straighten the arms."

This is an interesting case of strychnin poisoning, because it is most probably one of the earliest cases of poisoning by this drug in this country, or, perhaps, in any other where the evidence was conclusive of deliberate murder; moreover, the conviction was supported by the confession of the murderer, who was convicted and executed.

879.—Case XXXI. Poisoning of a Family by Barium Carbonate.

(Compiled by Assistant Professor Charles Harrington, M. D., of Harvard Medical School.)

(Vierteljahrsschrift für gericht. Med. und öffentl. Sanitätswesen, Bd. XXVIII. p. 248.)

On March 29, 1874, the family B., of Hamburg, celebrated the sixty-eighth birthday of the father. At five o'clock in the afternoon chocolate and cake were passed around. A piece of the cake was given to a canary and another to a dog. Five minutes afterwards the canary died without having shown any symptoms of disease. The dog was seized with vomiting and diarrhea, and on the next day seemed very sleepy. Within from about a quarter to a half an hour after eating the cake the family were seized with similar symptoms. Herr B., aged sixty-eight, Frau B., aged fifty-eight, Frl. B., aged nineteen, and Frl. R., a friend, aged seventeen. The first to be seized were the young ladies, who turned pale, and complained of a peculiar sensation of stretching of the skin. In a short time this was followed by violent vomiting and diarrhea. Shortly afterwards Frau B. was seized with the same symptoms; she described the sensation in the skin of the face to be as though she had washed in hot water. Herr B. was taken with general discomfort and rumbling in the abdomen. Meanwhile, they were visited by two persons, who suspected as a cause of the symptoms

the cake which already was half eaten. In consequence of their suspicions they declined to eat any, and remained well. Following their advice, the three ladies, who had already taken "Hoffmann's Drops" without effect, drank large quantities of milk; but Herr B. firmly refused to take any. After drinking the milk increased vomiting occurred, which, however, soon diminished. In the evening the ladies were better, though on the next day they felt weary and devoid of strength, and complained especially of weakness in the legs. Herr B., on the contrary, suffered during the whole evening, and not before 10 o'clock could he be persuaded by his wife to drink milk; violent vomiting followed. In the course of the night diarrhea set in: toward 4 o'clock in the morning, on going to the water-closet, he fell to the floor, and required the assistance of his wife and daughter to get back to bed on account of loss of power in his legs. Toward morning the vomiting ceased, but a constantly increasing paralysis from below upwards was developed. At 10 o'clock he was only able to nod his head. At two in the afternoon he was found by his physician with a congested face, bolstered up in a half-sitting posture in bed; voluntary motion of the arms and legs was quite impossible, and articulation was difficult. Sensation in the paralyzed parts was unimpaired, and reflex action was very manifest on tickling the feet. The sphincters were unaffected; the breathing was somewhat increased in frequency, and there were tracheal *râles;* the sensorium was affected. Pulse and temperature were normal; there were no pains even in the region of the stomach. In the evening, at 9 o'clock, twenty-eight hours after eating the cake, he died. He had eaten no more of the cake than any of the others who were seized.

The autopsy was deferred until sixty hours after death, when decomposition had made such progress that the results were quite negative. The only noticeable features were the prominent dark color of the gray substance of the brain, and the apparently complete absence of injury to the mucous membrane of the throat, œsophagus, stomach, and intestine.

In order to obtain more knowledge concerning the nature of the poison, the experiment of feeding birds with the cake was tried. A few minutes after eating they were seized with unsteadiness in the legs, and were soon unable to stand without falling. Flying then became impossible, and in a few minutes more the birds were lying dead. From several experiments it was noticed that the paralysis did not appear simultaneously on both sides; the birds always fell on the same side. A dog was given 20 grams of the cake, and in two and a half hours was seized with vomiting and diarrhea, but recovery followed.

The same symptoms occurred in birds and a dog which were fed on the flour from which the cake was made.

The analysis of the flour showed the presence of 10 per cent of carbonate of barium, and of 0.3 per cent of sulphate of barium; the cake yielded 2.74 per cent of the carbonate and of 0.43 per cent of the sulphate of barium. The contents of the stomach of the deceased, amounting to but 8 grams, yielded perceptible traces of the barium. At about the same time (April 6 and 7) it became known that several persons in two separate parties had become ill after eating cake. Both cakes came from the same confectioner, who obtained his flour at the same shop as the family B. Consequently the two specimens of cake were analyzed for barium, although two chemists had already pronounced them free from injurious substances. Carbonate of barium was found in both in very considerable amount. The flour from which all the cakes were made came from a shop where drugs, paints, and chemicals were sold. A wilful and malicious adulteration seemed improbable, while a careless admixture was more to be conjectured. It is probable that it had been the intent to adulterate the flour with chalk and that the barium had been accidentally substituted.

880.—SUMMARY OF RESULTS, GIVING CONCLUSIONS AS TO EFFECTS OF BORIC ACID AND BORAX ON DIGESTION AND HEALTH.

(Published August, 1904, by Dr. Wiley, Chief of Bureau of Chemistry, U. S. Department of Agriculture, and authorized to be republished in this work; the preliminary portion is omitted.)

Ratio of Food Consumed to Body Weight.

Of interest in connection with the other purposes of this investigation is a study of the relation of the weight of food consumed to the body weight, which was made in detail during the first series of observation. This study was made of each individual article of diet, and included a statement of the ratio of the weight of the food, including the water consumed, and the ratio of the weight of the dry matter in the food, to the body weight. During the fore period, first series of observations, the average daily weight of the moist food, including water drunk, was 4.20 per cent of the total weight of the body; during the preservative period, 4.22 per cent; and for the after period, 4.21 per cent. That is, in about twenty-four days, the average healthy young man would consume a quantity of moist food, including water drunk, equal to his own weight.

It is seen by the above that the administration of the preservative caused very little variation in the weight of food consumed compared with the weight of the body.

Reduced to water-free basis, the quantity of food consumed in relation to the weight of the body is as follows:

	Per cent.
Fore period	0.96
Preservation period	0.99
After period	1.01

These data show that there is very little difference between the total quantity of dry matter in the food during the three periods. The total quantity of dry matter in the food consumed daily is, in round numbers, 1 per cent of the weight of the body. For a man weighing 150 pounds, therefore, the quantity of dry matter daily consumed in the food is about 1.5 pounds. It is also interesting to note that the daily ratio of the moist food, including the water drunk, is a little more than four times as great as that of the dry food.

Similar data for the other series of observations are recorded; but the further discussion of the problem is not deemed necessary.

Influence of the Preservative upon the Weight of the Body.

In every series there was a marked tendency on the part of boric acid and borax to diminish slightly the weight of the body, although this tendency was in some instances checked during the after periods, and a portion of the loss of weight was regained. In general, however, there was a tendency to continue the loss of weight during the after periods.

Excretion of the Added Preservatives.

The borax and boric acid taken into the stomach during the progress of these experiments were excreted almost entirely by the kidneys. In the first series of experiments, 83.05 per cent were thus excreted, in the second series 82.85 per cent, in the third series 63.87 per cent, in the fourth series 82.96 per cent, and in the fifth series 75.17 per cent. During the course of observation 607.4 grams of preservative were given, either in the form of boric acid or the equivalent in borax, of which 468.69 grams were excreted in the urine, or 77.16 per cent of the whole. These numbers include the data for Series III., where the quantity of the preservative recovered in the urine appears to be abnormally low. In round numbers it may be said that 80 per cent of the boric acid and borax taken into the

system in foods is excreted in the urine. It is probable that the rest is chiefly excreted with the perspiration. Only small quantities are found in the feces.

Effect of Preservatives upon the Composition of the Feces.

A careful study of the effect of the preservative administered, upon the composition of the feces, shows a slight tendency to increase the amount of water therein. There is, however, no tendency of any marked nature, even when the preservatives are given in large quantities, to excite diarrhea. The administration of the preservative produces a slight increase in the weight of dry matter in the feces.

Influence of the Preservative upon the Metabolism of Nitrogen.

There is only a slight effect produced, as a whole, as determined by the data of experiment, upon the excretion of nitrogen. The individual variations are somewhat marked, showing the danger of depending too positively upon data from only one or two persons. A slight tendency is shown, however, on the part of the preservative, to decrease the excretion of nitrogen, which tendency becomes more marked after the withdrawal of the preservatives. For instance, the average nitrogen balance of the four series of observation (excluding Series II.) during the fore periods is 1.009, during the preservative periods 1.12, and during the after periods 1.74, grams per day. Expressed as a percentage, the combined data show an excretion of 94.2 per cent of nitrogen taken in the food during the fore periods, 93.6 per cent in the preservative periods, and 90.1 in the after periods.

The general summary of all the experiments with borax and boric acid indicates the largest elimination of nitrogen in the fore periods, an intermediate amount in the preservative periods, and the smallest elimination in the after periods.

This relation is either produced by causes other than the administration of the preservative, or the effect of the preservative continues after its administration has ceased, and even after the preservative itself has ceased to be excreted from the body. It is not impossible that such an influence may be exerted. The retarding influence of the preservative probably increases with the length of the experiment,—especially in those cases in which the amount of preservative administered is progressively increased. When the administration of the preservative is discontinued, the elimination of

nitrogen is probably at the lowest point (if depressed by the preservative), and yet, during the first days of the after period (at least while the preservative is still in the system), the amount of nitrogen eliminated is probably as low as on the preceding days. There may be a tendency of the preservative, in the large amounts in which it is administered, to increase the formation of difficultly soluble compounds of nitrogen, and by that means, if no other, to retard its elimination from the body.

The Effect of the Preservative upon the Metabolism of Phosphoric Acid.

A study of the data relative to the influence of boric acid and borax upon the metabolism of phosphorus reveals many contradictory results. When, however, all the data are collected into one expression, it is found that the influence of these bodies added to the food is distinctly marked on the metabolism of phosphorus and phosphoric acid. There is a distinct tendency shown by them to increase the quantity of phosphoric acid excreted during the period of the administration of the preservative. In the combined data of Series I., III., IV., and V., the average per cent of phosphoric acid, taken in the food, eliminated during the fore periods of observation is 97.3, during the preservative periods 103.1 per cent, and during the after periods 97.0 per cent.

Influence of the Preservative upon the Elimination of Fat.

The influence of boric acid and borax upon the metabolism of fat is not very marked. There is a slight tendency shown to decrease the elimination of fat in the feces during the administration of the preservative, and a tendency to recover is shown during the after periods. The percentage of fat, ingested in the food, eliminated during the fore periods is 4.1, during the preservative periods, 4.0 per cent, and during the after periods 4.2 per cent. These data show that almost no disturbance in the metabolism of fat is caused by the administration of the preservative.

Influence of Boric Acid and Borax upon the Oxidation of the Combustible Matter in the Food.

collected data of all the series (except Series II.) show that

6.4 per cent of the combustible matter in the food is eliminated, unburned, during the fore periods, 6.6 per cent during the preservative periods, and 7.0 per cent during the after periods. These data show a slight tendency on the part of the preservative to interfere with the combustion of the food in the body, and this tendency is continued in even a more marked manner during the after periods.

Influence of the Preservative upon the Solids Excreted.

The solids' summary for all of the series (except Series II.) shows that the average quantity of solids in the food during the fore periods is 631.5 grams, during the preservative periods 627.6 grams, and during the after periods 614.1 grams. The average daily quantity of solids appearing in the feces in the fore periods is 25.6 grams, in the preservative periods 28.6 grams, and in the after periods 28.3 grams. The average quantity appearing in the urine during the fore periods is 64.48 grams, during the preservative periods 59.37 grams, and in the after periods 56.20 grams. The average balance of total solids during the fore periods is 544.701 grams, during the preservative periods 539.875 grams, and during the after periods 530.123 grams. These data show a marked tendency on the part of the preservative to increase the total solids excreted in the feces, and to decrease the total solids excreted by the urine. There is a distinct tendency manifested by the preservative to interfere with the processes of digestion and absorption. Inasmuch, however, as the total quantity of solids administered in the food varied slightly in the different periods, a fairer interpretation is obtained by comparing the percentages of the total solids exhibited in the food eliminated by the feces and urine respectively. In this comparison it is found that the total percentage of solids in the food eliminated in the feces during the fore periods is 4.1, during the preservative periods 4.6, and during the after periods 4.6. The percentage of solids in the food eliminated in the urine during the fore periods is 10.2, during the preservative periods 9.5, and during the after periods 9.1. These percentages indicate also very strongly the influence exerted by the preservative mentioned above. It must be remembered, also, in this connection, that practically 80 per cent of the preservative administered is recovered in the urine, increasing to that extent the total solids thus eliminated. In spite of this, however, there is a marked decrease in the total solids in the urine, and a marked increase in the total solids in the feces.

Effect of Boric Acid and Borax upon the Urine.

Elimination of nitrogen.—The combined data of the four series (excluding Series II.) show that the percentage of nitrogen, ingested in the food, eliminated in the urine during the fore periods is 85.7, during the preservative periods 85.1, and during the after periods 81.1. This shows a tendency on the part of the preservative to diminish the percentage of nitrogen excreted in the urine, and this tendency is continued in a very marked manner in the after periods.

Reaction.—The data of Series II., III., and V., show a marked tendency on the part of boric acid to increase the acidity of the urine. In no case during the administration of boric acid was an alkaline reaction observed. In the case of the urine, the marked acidity imparted to it by boric acid is continued in most cases throughout the after periods. The data of Series IV. and V., on the contrary, show a marked tendency on the part of borax to diminish the acidity of the urine, and in several instances this substance imparted to the urine an alkaline reaction. These facts indicate that a large part of the borax and boric acid administered is excreted unchanged in chemical composition.

Quantity.—Very little effect is produced by these preservatives upon the volume of urine, although there is a slight tendency manifest to decrease the amount. There is a slight tendency also manifested during the administration of the preservatives to decrease the total solids in the urine. In this connection, however, it must be considered that the season of the year has a marked effect upon the amount of urine secreted, the tendency being to secrete larger quantities in cold weather than in warm. Combining the data of Series I., III., IV., and V. for those members completing the series, we find that the average daily amount of urine secreted during the fore periods, per individual, is 969 cubic centimeters, during the preservative periods 960 cubic centimeters, and during the after periods 952 cubic centimeters. These data show almost no effect of the preservatives on the quantity of urine secreted; but there seems to be a slight tendency to decrease the amount secreted in the preservative and after periods.

Albumin.—In those few cases where there was normally a mere trace of albumin in the urine, it is shown by the data that the general tendency of the preservative used is to increase the trace of albumin in the urine, and this increase is manifested also during the after periods.

Microscopic bodies.—Microscopical examinations of the urine were made for the following substances.

Uric acid crystals; urates; oxalate of lime; phosphates, (a) crystalline phosphates, (b) amorphous phosphates; epithelium cells of all kinds; leucocytes; red blood cells; casts, (a) hyaline, (b) finely granular, (c) coarsely granular, (d) epithelial, (e) other forms; mucous cylindroids; mucous strands.

The microscopic examinations were made at three periods during each series except in Series I., during which time the microscopic supervision of the urine had not been instituted. The examinations were made once during the fore period, once or more during the preservative period, and once near the close of the after period.

Reviewing the data as a whole, in regard to the appearance of these microscopical bodies in the urine, the facts which appear prominently are the great variations in the number and character of these micro-chemical bodies. They occur constantly in some cases in very much greater abundance than in others. There are a few cases, in fact, quite a number, where the relative abundance of these bodies seems to be increased during the administration of the preservative. There is a smaller number of cases in which the contrary fact occurs. In the greater number of cases, however, the administration of the preservative appears to have had no influence upon the relative abundance of these bodies. The data, therefore, as a whole, cannot be regarded as conclusive respecting the influence of the preservative upon the number or kind of micro-chemical bodies occurring in the urine.

The Effect of the Preservative upon the Number of Corpuscles and upon the Hemoglobin in the Blood.

There was no regular influence established relating to the effect of the preservative in increasing or decreasing the number of corpuscles in the blood. The data in individual cases are often contradictory, and a general summary of them leads to no conclusive result. The final deduction can only be drawn that, if the preservative affects the number of corpuscles and the amount of hemoglobin at all, it does so in very irregular manner, differing in different individuals, and in a way which cannot be used as a basis of any definite conclusion.

CONCLUSIONS.

Necessity of Mineral Substances in the Blood.

In the consideration of the action of preservatives of a mineral na-

ture, such as borax and boric acid, it must be remembered that the animal, as well as the plant, possesses a certain mineral hunger. In other words, mineral substances play a double rôle in animal and plant nutrition. First, they may serve as real foods, necessary to the formation and nutrition of the tissues. In the second place, they are necessary to the functional activity of the various organs of the body, irrespective of any part they may take in direct nutrition.

The necessity of saline solutions in the blood is known to every physician and physiologist. If the blood were deprived of all of its saline constituents, the circulation would be impeded, restricted, or stopped, and death would result. In cases of collapse in disease, saline injections in the blood are often used as a restorative measure. These salts, in solution, stimulate the heart's action, and undoubtedly are active in the osmotic operations of the cells. This is one of the facts which show the intimate relation existing between physical chemistry and physiology.

Common salt is the most frequent and most abundant of the saline constituents of the blood; but the alkalinity of the blood is not due, of course, to common salt, which is a neutral substance. The existence of alkaline carbonates or other alkaline salts is necessary to the vital functions. While it is true that the digestion in the stomach takes place in an acid solution, it is likewise true that any excessive acid must be neutralized, and enough of alkali added in the small intestine, in order that the further digestion of the food may properly take place. That saline bodies other than common salt or the alkaline carbonates may be useful, however, in the performance of the vital functions cannot be denied, though it might be difficult to demonstrate their absolute necessity. Hence the introduction of saline bodies, which may or may not be of an antiseptic character, may, within certain limits, have a favorable influence upon health and digestion. At the same time it should not be forgotten that all excess of such bodies imposes upon the excretory organs an additional burden, which, while it might not impair their efficiency even for a number of years, might finally produce a condition of exhaustion which would be followed by serious consequences. Especially is this remark true of the kidneys, which appear to be a general clearing house for all the surplus of saline matters ingested in the foods.

Are Minimal Quantities of Preservatives Permissible?

It is admitted by all who have examined the subject in a critical

way, even by the users of preservatives, that, in certain maximum quantities, the limit of toleration is reached in each individual, and positive injury is done. But it is also well recognized that many, if not all, of the usual foods, when used in large excess, produce injurious results. The many cases of disease produced by overeating, or by eating improperly prepared or poorly cooked food, or by eating at unusual times, are illustrations of this fact. Upon this basis, and upon the further statement that, when used in extremely small quantities, the preservatives in question cannot be regarded as harmful, is founded the principal argument in favor of the use of the preservatives, aside from the fact that the foods themselves are kept in a better and more wholesome state.

It is only proper to give to this argument full consideration, and not to brush it aside as illogical and irrelevant. It is evident that any attempt to determine experimentally the effect of extremely minute quantities of any preservative, even when used continuously, would not be likely to lead to any definite result. In the foregoing data we have illustrations of the fact that even large quantities of the preservative employed—larger by far than would probably ever be found in any food product—do not always act in such a way as to permit of definite interpretation. The claim, therefore, that the use of such preservatives is justified when the amount is extremely small, and when even these small amounts are used only at intervals, and not continuously, is worthy of careful consideration.

An illustration which is pertinent may be taken from the particular preservatives with which the foregoing experiments have been made, namely, boric acid and borax. One of the food products to which these preservatives are very commonly added is butter. This statement should not be taken to imply that, in butter prepared for domestic use in this country, borax is found to any considerable extent. When butter, however, is to be transported over long distances, and necessarily kept a long while, the addition of borax is very frequently practised.

The dietetic data, which have been accumulated in the course of this experiment, show that the quantity of butter consumed daily varies from 30 to 70 grams. Suppose, as a maximum, we say that the quantity of butter consumed in any one case, daily, is 100 grams, and that it contains 1 gram of boric acid, or an amount of borax equivalent thereto. The maximum quantity of boric acid used in a day in this case would be 1 gram. In point of fact, however, it would rarely, if ever, reach this amount; but, even in those cases

where butter is eaten freely, probably ½ gram would be about the maximum quantity consumed. Further than this, 1 per cent of boric acid, or its equivalent in borax, in butter is a very large quantity. Probably, as a rule, not more than ½ of 1 per cent is employed. In this case the quantity of boric acid likely to be consumed by any one individual in a day would be reduced to ¼ of a gram.

In the case of meats preserved by borax, although larger quantities are eaten than of butter, it is not likely that any larger quanties of borax would be consumed. Thus it appears that those who habitually eat butter and meat preserved with borax might be consuming half a gram, or a little more, of boric acid per day. But preserved meats are not regularly eaten, and hence the quantity mentioned is likely to be overestimated. It would be unwise to affirm, in a case of this kind, in the light of the data obtained by the experiments, that such a minimum consumption of borax, and especially when not a continuous one, would prove deleterious within any reasonable time of observation. The question then arises, Does the absence of such proof, or the impracticability of obtaining it, serve as a justifiable excuse for the use of this preservative?

This question ought not to be decided alone, because the principle of the decision must stand, not only for boric acid and borax, but for every preservative used in foods. In other words, whatever principle is established for judgment as to the use of boric acid in small portions must also be applied to the use of every other preservative used in foods. The principle must also be still further established so that whatever may be established as regards butter or meat must be admitted in respect of every other substance used in food. Hence, before admitting the full force of the argument based on minimal quantities, the full significance of such an admission must be considered, and the practically unlimited extent of its application acknowledged.

This leads to the discussion of the fact that in the majority of cases the labor of freeing the system from added preservatives falls principally upon the kidneys. In the method of life in vogue in this country the kidneys are already hard-worked organs. Americans probably eat more freely than the citizens of almost any other country, with the possible exception of England. Large quantities of nitrogenous foods are consumed. In the breaking down of the nitrogenous tissues the kidneys are the chief organs for the excretion of the *débris*. The addition of any further burden, therefore, no mat-

ter how minute, is to be deplored. If, however, the principle be admitted that injurious substances may be used in such small quantities as to be practically harmless, then we find the way open for loading upon the kidneys many different functions in addition to those which they now discharge. If they may be justly called upon to eliminate the small quantities of boric acid added in food, they cannot logically be freed from the necessity of eliminating also minute quantities of salicylic acid, saccharin, sulphurous acids, and sulphites, together with the whole list of the remaining preservatives, which are eliminated principally through the kidneys. It would be useless to contend that the occasional consumption of small quantities of boric acid in a sausage, in butter, or in preserved meat, would produce, even upon delicate stomachs, any continuing deleterious effect which could be detected by any of the means at our disposal; but, naturally, it seems that this admission does not in any way justify the indiscriminate use of this preservative in food products, implying, as it would, the equal right of all other preservatives of a like character to exist in food products, without restriction.

It appears, therefore, that there is no convincing force in the argument for the use of small quantities, unless it can be established that there is only a single preservative used in foods, that this preservative is used in only a few foods, that it will be consumed in extremely minute quantities, and that the foods in which it is found are consumed at irregular intervals and in small quantities. On the other hand, the logical conclusion, which seems to follow from the data at our disposal, is that boric acid, and equivalent amounts of borax in certain quantities, should be restricted to those cases where the necessity therefor is clearly manifest, and where it is demonstrable that other methods of food preservation are not applicable, and that, without the use of such a preservative, the deleterious effects produced by the foods themselves, by reason of decomposition, would be far greater than could possibly come from the use of the preservative in minimum quantities. In these cases it would also follow, apparently, as a matter of public information, and especially for the protection of the young, the sick, and the debilitated, that each article of food should be plainly labeled and branded in regard to the character and quantity of the preservative employed.

Effect of Boric Acid and Borax upon General Health.

The most interesting of the observations which were made during the progress of the experiments was in the study of the direct ef-

fect of boric acid and borax, when administered in food, upon the health and digestion. When boric acid, or its equivalent in borax, is taken into the food in small quantities, not exceeding half a gram (7½ gr.) a day, no notable effects are immediately produced. The medical symptoms of the cases in long-continued exhibitions of small doses, or in large doses extending over a shorter period, show, in many instances, a manifest tendency to diminish the appetite, and to produce a feeling of fullness and uneasiness in the stomach, which, in some cases, results in nausea, with a very general tendency to produce a sense of fullness in the head, which is often manifested as a dull and persistent headache. In addition to the uneasiness produced in the region of the stomach, there appear, in some instances, sharp and well-located pains, which, however, are not persistent. Although the depression in the weight of the body, and some of the other symptoms produced, persist in the after periods, there is a uniform tendency manifested, after the withdrawal of the preservative, toward the removal of the unpleasant sensations in the stomach and head above mentioned.

The administration of boric acid to the amount of 4 or 5 grams per day, or borax equivalent thereto, continued for some time, results in most cases in loss of appetite, and inability to perform work of any kind. In many cases the person becomes ill and unfit for duty. Four grams per day may be regarded, then, as the limit of exhibition beyond which the normal man may not go. The administration of 3 grams per day produced the same symptoms in many cases, although it appeared that a majority of the men under observation were able to take 3 grams a day for a somewhat protracted period, and still perform their duties. They commonly felt injurious effects from the dose, however, and it is certain that the normal man could not long continue to receive 3 grams per day.

In many cases the same results, though less marked, follow the administration of borax to the extent of 2 grams, and even of 1 gram per day, although the illness following the administration of borax and boric acid in those proportions may be explained in some cases by other causes, chiefly grippe.

The administration of borax and boric acid to the extent of ½ gram per day yielded results markedly different from those obtained with larger quantities of the preservatives. This experiment, Series V., conducted as it was for a period of fifty days, was a rather severe test, and it appeared that in some instances a somewhat unfavorable result attended its use. On the whole, the results show

that ½ gram per day is too much for the normal man to receive regularly. On the other hand, it is evident that the normal man can receive ½ gram per day of boric acid, or of borax expressed in terms of boric acid, for a limited period of time, without much danger of impairment of health.

It is, of course, not to be denied that both borax and boric acid are recognized as valuable remedies in medicine. There are certain diseases in which these remedies are regularly prescribed, both for internal and external use. The value which they possess in these cases does not seem to have any relation to their use in the healthy organism, except when properly prescribed as prophylactics. The fact that any remedy is useful in disease does not appear to logically warrant its use at any other time.

It appears, therefore, that both boric acid and borax, when continuously administered in small doses for a long period, or when given in large quantities for a short period, create disturbances of appetite, of digestion, and of health.

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